A PROSPECTIVE STUDY ON

CYSTOID MACULAR EDEMA

FOLLOWING CATARACT SURGERY

Dissertation Submitted for

M.S.Degree(Branch III) Ophthalmology

April 2013.

THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY

CHENNAI

Dept. Of Ophthalmology

Govt. Rajaji Hospital

Madurai

CERTIFICATE

This is to certify that this dissertation entitled "**A PROSPECTIVE STUDY ON CYSTOID MACULAR EDEMA FOLLOWING CATARACT SURGERY**" has been done under my guidance in the Department of OPHTHALMOLOGY, MADURAI MEDICAL COLLEGE, MADURAI.

I Certify regarding the authenticity of the work done to prepare this dissertation.

Dr.P.THIYAGARJAN , M.S.,D.O.,
PROFESSOR & H.O.D
Dept. Of Ophthalmology
GOVT. RAJAJI HOSPITAL
MADURAI MEDICAL COLLEGE,
MADURAI.

DECLARATION

I, **Dr. A.VIJAYALAKSHMI**, Solemnly declare that the dissertation titled, "**A PROSPECTIVE STUDY ON CYSTOID MACULAR EDEMA FOLLOWING CATARACT SURGERY"** has been prepared by me.

This is submitted to the "**THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY, CHENNAI**, In partial fulfillment of the requirement for the award of M.S., (Ophthalmology) Branch-III degree examination to be held in **APRIL 2013.**

Place: Madurai

Date: **Dr. A. VIJAYALAKSHMI**

ACKNOWLEDGEMENT

I am grateful to the Dean, Madurai Medical College and Govt.Rajaji Hospital, Madurai for permitting me to utilize the clinical materials of this hospital.

I am extremely grateful to ***Dr.P.THIYAGARAJAN, M.S.D.O.,*** Professor and Head of the Department of Ophthalmology, Madurai Medical College, Madurai, for his guidance and help for executing my study.

. I am extremely indebted to my beloved guide ***Dr.G.SRINIVASAN, M.S.,D.O.***, Professor of ophthalmology, Madurai Medical College, Madurai for his constant encouragement and guidance throughout this dissertation.

I am grateful to ***Dr.A.R.ANBARASI, M.S.,D.O.,*** Assistant Professor of ophthalmology for her valuable guidance, support and encouragement rendered to me during the study.

My sincere thanks to all my Assistant Professors for their valuable suggestions in carrying out this study.

I thank my study subjects, who formed the back bone of this study and without whom this work would not have been possible.

Last but not the least, I thank "God the Almighty", for being my guiding light all the way.

CONTENTS

PART-1

12. Management

-Preventive Measures & Treatment

13. Complications

14. Review of Literature

PART - 2

15. Aims and Objectives

16. Materials and methodology

17. Tables and graphs

18. Discussion

19. Summary

20. Conclusion

21. Bibliography

22. Proforma

23. Master chart

ABBREVIATIONS

ACIOL - Anterior Chamber Intraocular Lens

BCVA - Best Corrected Visual Acuity

BRB - Blood Retinal Barrier

CME - Cystoid Macular Edema

DM - Diabetes Mellitus

ERG - Eletroretinogram

ECCE - Extra Capsular Cataract Extraction

FFA - Fundus Fluorescein Angiography

HT - Hypertension

ICCE - Intra Capsular Cataract Extraction

INL - Inner Nuclear Layer

IOP - Intra Ocular Pressure

IOL - Intra Ocular Lens

Nd YAG - Neodymium Yttrium Aluminium Garnet

NSAIDS - Non Steroidal Anti Inflammatory Drugs

OCT - Optical Coherence Tomography

OPL - Outer Plexiform Layer

ONH - Optic Nerve Head

PCR - Posterior Capsular Rupture

PCIOL - Posterior Chamber Intra Ocular Lens

PST - Posterior Sub Tenon

PHACO - Phacoemulsification

PMMA - Poly Methyl Methacrylate

PPV - Pars Plana Vitrectomy

RTA - Retinal Thickness Analyser

SLO - Scanning Laser Ophthalmoscopy

SICS - Small Incision Cataract Surgery

UV - Ultraviolet

INTRODUCTION

Cataract represents global public health challenge for all countries. It still remains as the major cause of preventable blindness. Cataract surgery is the commonest surgery performed in the field of ophthalmology.

Among different surgical techniques performed for cataract, extracapsular cataract extraction (ECCE) / small incision cataract(SICS) surgery / Phacoemulsification with posterior chamber intraocular lens implantation is the commonest one.

Despite these advantages, significant complication that can lead to visual impairment is cystoid macular edema. If steps are not taken properly to prevent, identify & treat cystoid macular edema, it can be considered as 2nd most common cause of preventable blindness next to cataract.

ANATOMY OF NORMAL MACULA

Macula lutea is situated in posterior pole, centre of macula is about 3 mm temporal to optic disc and 1 mm inferior to optic nerve head. Macula is approximately a circle with radius of 2.75 mm centered at fovea. Beginning at the centre, foveola is about 0.35mm diameter. In this region, rod cone ratio is 1:2. Its base is about 0.1mm diameter which is free of cells except outer segment of photoreceptors.

Fovea is 1.5mm diameter. Nerve fiber layer, ganglion cells, inner nuclear layer, inner plexiform layer are absent at fovea. Thickness of retina is 0.25 mm at the fovea. Fovea contains 10%of cones of whole retina. Cone density remains constant outside this region. Rods are absent in central 0.25mm of fovea. Capillary free zone is 0.4mm in diameter centered at fovea.

Parafoveal zone is an area measuring 0.5mm surrounding fovea. Rod cone ratio is 1:1 in this region. Rest of area is known as perifoveal zone. Macula lutea or yellow spot extends about 1mm laterally & 0.8mm above and below.

At the macula, ganglion cells are much more numerous than elsewhere in retina being arranged in several layers. Outer plexiform layer is also thicker than elsewhere, referred to as Henle's layer. Also, there is progressive disappearance of

rods which are replaced by cones. Pigment epithelium layer & choriocapillaries are thicker at the macula which is significant because macula has no blood vessels.

Cone nuclei are heaped up in the fovea centralis but the remaining retinal layers are displaced laterally so that the internal limiting membrane which is very thin lies directly on the receptor nuclei. In the surrounding retina, nuclei of inner nuclear layer and ganglion layer increases in number, the volume contributing to raised rim of foveal depression.

In retina, each ganglion cell is connected to many visual cells, upto 100 rods, whereas each cone is connected to only one ganglion cell. According to Wolffe, outer plexiform layer is made up of arborization of axons of the rods and cones with dendrites of bipolar cells. This layer also includes muller's fiber and processes of horizontal cells. Elsewhere in the retina, this layer has a reticular structure, but takes a fibrous structure as macula is approached and is called Henle fiber layer. Fibers run vertically, then obliquely near the macula and finally parallel to the surface. This layer is thickest in the macula, but absent in fovea. This arrangement leads to accumulation of fluid in typical radiating petalloid pattern.

Muller fibres are long complicated structures traversing the entire thickness of the retina from internal limiting membrane to external limiting membrane.

Nucleus of Muller fibre is bipolar and is situated at the level of inner nuclear layer.

Floor of foveal center is rich in Muller fibres.

DEFINITION OF CYSTOID MACULAR EDEMA

Under normal conditions, internal and external blood retinal barriers separate retina from plasma by a system of restricted permeability. This ensures maintinenance of homoestasis and a consistent internal milieu in the retina. When these barriers are disrupted due to any cause, there is increased entry of plasma constituents into extracellular space of the retina. Entry of plasma proteins and water causes expansion of the extracellular space, often associated with accumulation of fluid in the macular area which is called macular edema.

Macular edema may develop in inflammatory, occlusive, degenerative, infiltrative, traumatic and toxic condition of retina, uveal tract, vitreous or even in some systemic diseases.

Fluid accumulation in radially arranged fluid spaces localized to outer plexiform and inner nuclear layer of the para foveal retina is termed as cystoid macular edema (CME)

ETIOLOGY OF CYSTOID MACULAR EDEMA

1. Associated with intraocular surgical procedure
    a. Cataract surgery with or without IOL implantation **(Irvine Gass syndrome)**
    b. Cataract surgery with complications e.g., vitreous loss
    c. Aphakic keratoplasty
    d. Vitreous surgery
    e. Retinal surgery e.g, scleral buckling
    f. Retinal photocoagulation or cryotherapy
    g. Post surgical hypotony
2. Associated with systemic and retinal vascular diseases
    a. Retinal vein occlusion
    b. Retinal telengiectasia e.g.,coats disease, macular telengiectasia
    c. Radiation retinopathy
    d. Hypertensive retinopathy
3. Associated with diabetes mellitus
4. Associated with choroidal and retinal pigment epithelial disease
    a. Choroidal hemangioma

b. Malignant melanoma

c. Age related macular degeneration

d. Retinitis pigmentosa

e. Retinal pigment epitheliopathy

f. Pseudo vitelliform dystrophy

5. Associated with trauma or traction

a. Idiopathic preretinal fibrosis

b. Cellophane maculopathy

c. Blunt ocular trauma

d. Electric injuries to retina

e. Solar retinopathy

6. Associated with pathology of the vitreous

a. Vitreous loss

b. Vitreous adhesions to the wound

7. Associated with uveitis

a. Anterior uveitis e.g., non granulomatous iridocyclitis

b. Intermediate uveitis or pars planitis

c. Posterior uveitis e.g., behcet's disease

8. Associated with drugs and pharmacological agents
    a. Nicotinic acid
    b. Topical epinephrine in aphakic eyes
    c. Oral contraceptive pills
    d. Hydrochlorthiazide
    e. Latanoprost, betoxolol, timolol

CLASSIFICATION OF CYSTOID MACULAR EDEMA

1. **Based on visual acuity**

   a. Angiographic CME

   Refers to typical petalloid pattern of macular fluorescent leakage seen with fluorescent angiography but is not associated with decreased visual acuity.

   b. Clinical CME

   It is associated with same macular pattern of fluorescent leakage on angiography with visual symptoms and a decrease in visual acuity of atleast two snellen lines or visual acuity worse than 20/40.

2. **Based on severity of clinical course**

a. **Acute CME** resolves spontaneously in majority of patients in six months following surgery.

b. **Chronic CME** when it persists for longer than six months, spontaneous resolution is less likely and is termed as chronic CME.

INCIDENCE

Despite major advances in cataract surgery techniques and instrumentation, CME[1,2] continues to be one of the leading causes of decreased visual acuity after cataract surgery. The prevelance of CME after cataract surgery depends upon experience of the surgeon, type of surgery, design of study, definition of disease , method for diagnosis, patient population, prior ocular surgery, coexisting ocular diseases, accompanying systemic diseases, surgical complications and follow up criteria.

Angiographic CME is very common and has been reported to occur in over 50% of patients after cataract surgery with or without IOL implantation. But reduced visual acuity due to clinical CME occurs in upto 8% of patients. Incidence of pseudophakic CME also depends upon time lapse after surgery. It is highest 44% in first six weeks postoperatively, 24% at six months and 12% at 12 months. It has been rarely reported after one year [3].

PATHOPHYSIOLOGY AND ETIOPATHOGENESIS

Although it still not clear, why fluid accumulates preferentially in foveal region despite widespread distribution of inflammatory mediators, several distinguishing features of the macula are thought to play a vital role. High metabolic activity, decreased fluid drainage, thin internal limiting membrane with less prevention of diffusion of inflammatory mediators, the thickness and horizontal course of outer plexiform layer providing a reservoir for fluid accumulation[4].

BREAKDOWN IN BLOOD AQUEOUS BARRIER

Retina has barriers at 2 levels- retinal pigment epithelium and endothelium of retinal blood vessels. They have non leaking tight junctions. The blood aqueous barrier similarly has two barriers-tight junction of capillary endothelium in iris blood vessels and posterior uveal lining of non pigmented epithelium.

The integrity of blood aqueous and blood retinal barrier can be assessed by slit lamp biomicroscopy (leakage of proteins and cells) or by fluorophotometry[5,6] (fluorescein concentration across these barriers into vitreous and aqueous).

Breakdown in blood aqueous barrier has been implicated as the prime cause of psuedophakic CME. There is breakdown in the functional complexes of the perifoveal capillaries allowing leakage of fluid and resultant edema. Miyake[7] suggested that the occurrence of CME is related to synthesis of prostaglandin and other mediators. Surgical trauma leads to release of prostaglandins which causes breakdown in the blood aqueous barrier with resultant release of toxins and immune complexes.

Prostaglandins pass through the vitreous into posterior chamber and result in outpouring of serous fluid in the henle's layer. Increased levels of PGE& PGF have been demonstrated in patients with CME undergoing vitrectomy.

Therefore NSAIDS which inhibit the production of prostaglandin by blocking cyclooxygenase cycle are helpful in preventing as well as resolving CME after cataract surgery.

CHANGES IN THE VITREOUS

Changes in vitreous in the postoperative period may also play a role in etiopathogenesis. Vitreous cortex and vitreous face may serve as barriers for the diffusion of chemical mediators. However in cases with vitreous loss, anterior displacement of vitreous and increased vitreous liquefaction occurs. Even in

uneventful surgery, during which no vitreous loss occurs, anterior displacement of it can occur after cataract surgery.

Anterior displacement of the vitreous causes secondary changes in perifoveal vitreo-retinal interface, particularly in eyes in which spontaneous separation of the posterior hyaloid membrane has not occurred, playing a role in subsequent CME formation[8].

Vitreous incarceration may also play a role in CME formation by causing mechanical vitreo-retinal traction on the perifoveal retina, thus changing the vascular permeability of perifoveal retinal capillaries, leading to pooling of fluid in outer plexiform layer of retina.

HISTOPATHOLOGY OF CME

Histopathological studies[9] of eyes with CME have shown accumulation of water and proteinaceous materials causing marked swelling and edema with cystoid changes particularly of outer plexiform (henle's) layer and inner nuclear layer.

Electron microscopic studies of CME showed marked degeneration & swelling of muller cells with no enlargement of extracellular spaces which was described by Fine and Brucker[10]. Gass and coworkers found polycystoid expansions of the extracellular spaces that correspond to the well developed pattern on CME on angiography.

Increased destruction of muller cell cytoplasm and necrosis of adjacent cells leads to formation of larger cystic spaces. Loss of photoreceptors is a consistent finding.

Wolter[11] reported swelling of retina with formation of retinal folds, cystoid spaces in outer plexiform layer and inner nuclear layer and localized detachment of centre of macula in the area of external limiting membrane. In severe cases intraretinal hemorrhage with blood filled cysts may also occur.

ELECTRON MICROSCOPY

Fine and Bucker whose patients did not have well developed cystoid spaces on angiography found marked degeneration and swelling of the muller cells with no enlargement of extracellular spaces. Gass and coworkers found the polycystoid expansion of extracellular spaces that correspond to the well developed pattern of CME on angiography.

CLINICAL FEATURES OF CME

SYMPTOMS

1. **Asymptomatic**

Angiographic PCME (psuedophakic cystoid macular edema) is usually subclinical and transient . Majority of patients belong to this category.

2. **Impairment of vision**

Generally occurs 1 to 4 months after cataract surgery. There is mild to moderate drop in visual acuity. This visual acuity is usually about 6/18 or better and seldom drop to less than 6/60. In patients with chronic CME, there is persistent visual loss. Reduced central vision, metamorphosia, micropsia may be present.

3. **Loss of contrast sensitivity**

Reduction in contrast sensitivity[12] may occur in the presence of better visual acuity.

4. **Acute delayed drop in vision**

Rarely in cases with delayed PCME, patients may typically notice a sudden drop in their best corrected visual acuity by 2 to 3 lines on the snellen chart.

5. **Symptoms of intraocular inflammation**

Rarely patients present with symptoms of uveitis like pain, photophobia and an irritable eye.

SIGNS

Clinical signs of CME are often mild and may be unnoticed unless there is a higher index of suspicion. Eye often appears normal as the anterior segment signs are very subtle.

1. **Mild ciliary flush and iritis**

There may be mild ciliary flush along with iritis as is evidenced by flare, cells and mild anterior uveitis.

2. **Vitreous disturbance**

A ruptured posterior capsule, cells in the anterior hyaloid may be present.

3. **Malpositioned IOL**

This may cause pupillary distortion, iris tucking and iris chaffing.

4. **Fundus examination with I/O or 3 mirror contact lens**

It reveals thickening and edema of the macula with honeycomb appearance due to the presence of microcystoid spaces. The cysts are bigger centrally with teardrop shape. They are best seen with retroillumination on the slit lamp. Foveal reflex is absent or distorted and cells may be seen in posterior vitreous.

RISK FACTORS FOR CME

Absolute :

1. Iris incarceration in wound
2. Vitreous incarceration in wound
3. Postoperative uveitis
4. Postoperative vitritis
5. Vitreous in anterior chamber
6. Peaked pupil
7. Posterior capsular rent
8. Vitreous loss
9. Post Nd YAG capsulotomy

Relative:

1. Young patients
2. CME in fellow eye
3. Diabetes mellitus
4. Systemic diseases

Absolute risk factors

1. **Iris incarceration in wound**[13]

Uveal tissue incarceration in the incisional site can result in chronic PCME. This condition often leads to persistent low grade iritis. This may be responsible for a prolonged breakdown in the blood aqueous barrier and thereby PCME.

2. **Iris IOL contact**

This may occur with the ciliary sulcus fixated IOL especially 10 degree angulated ones. PMMA haptics are more prone for uveal irritation. Malpositioned IOL's can give rise to chronic uveal lens contact by causing pupillary distortion, iris tucking, iris chaffing, etc. This iris chaffing and capillary leakage result in breakdown of the blood aqueous barrier for long periods. Modern "in the bag" IOL's which have no contact with iris have minimized CME as they produce least trauma to blood aqueous barrier[14].

3. **Posterior capsular rupture**

Posterior capsular rupture especially if accompanied by vitreous loss has a greater predisposition for PCME[15]. This again is related to breakdown in the blood aqueous barrier and chronic inflammatory reaction propagated by this

complication. Primary posterior capsulotomy performed during ECCE carried a significantly increased risk of CME[16] than ECCE with intact posterior capsule. Also, angiographic PCME was found to be more common in primary post capsulotomy group than in those with intact capsule. Chronic uveitis set up by vitreous chaffing of the iris may be responsible in cases with associated vitreous disturbance. Thus it is evident that protective effect of posterior capsule is significantly negated by opening it.

4. Postoperative uveitis

This is important cause of PCME. Uveitis may be overt and marked with severe anterior chamber reaction. Commonly the eye may be apparently normal but careful examination often reveals mild ciliary flush, few cells and flare in anterior chamber. Varying degrees of flare have been demonstrated by LASER flaremeter in cases of CME[17]**.** All complications associated with the risk of postoperative uveitis also increase the risk of PCME. These include excessive manipulation during intraocular surgery posterior capsular rent, vitreous loss, preexisting uveitis, preoperative pilocarpine usage , hypermature leaking cataract ,uveal incarceration ,IOL manipulation. Inflammatory mediators like PG'S & leukotrienes play a key role in such inflammation.

5. Phtotoxicity and operative microscope

Photic injuries have been described after surgical exposure to excessive illumination of the operative microscope. Byrnes GA et al [18] reported 10% incidence of such injuries. Spectral analysis of light source of the operating microscope have shown that measurable amount of infrared and ultraviolet light more of it being in the 500- 700nm range.

6. Postoperative exposure to ultraviolet radiation

Besides photic injury during surgery, psuedophakic eye is also left unprotected from the external ultraviolet radiation after surgery. Crystalline lens absorbs most of incident UV radiation, thereby protecting the retina from a significant potential source of photic damage. This protection is lost when lens is removed. Implantation of IOL has ultraviolet absorbing chromophobes incorporated in PMMA substance can possibly restore this protection. Thus a UV absorbing IOL besides restoring normal spectral sensitivity and reducing erythropsia, stabilizes blood vitreous barrier and reduces incidence of CME [19].

Probable risk factors

1. Age

Younger patients are more prone to develop CME[20]. Reason for this is more active immune system in young which may be responsible in initiating the immune response to CME.

2. CME in the fellow eye

The incidence of clinically significant CME in the other eye when one eye has had this complication is reported to be high. Thus in a particular patients with CME, the second eye is considered as a prone eye. Prophylactic measures preoperatively (steroids or NSAIDS) have been recommended in such eyes [21].

3. Diabetes mellitus

Diabetics seem more prone to CME. Incidence of clinical CME among diabetics is reported as 9.3% and it's average period of presentation to be 3 to 6 months after surgery[22]. Duration of diabetes, its severity, grade of retinopathy, general complications are important factors in the development of CME in diabetics.

4. Systemic diseases

Patients with hypertension, cardiac disease, vascular diathesis, rosacea, telengiectasia, chronic alcoholism are reported to be at risk for developing CME [20].

INVESTIGATIONS

Slit lamp examination with contact or noncontact lens makes it possible to detect presence of retinal thickening, whether localized or extending to posterior pole. Use of narrow slit beam is useful in detecting cystoid spaces. Clinical suspicion of macular edema can be confirmed with the aid of variety of investigations. Tests may be grouped into 3 categories according to whether one is analyzing the basic underlying pathogenesis, effect of macular edema on retina or its impact on visual function.

A. Tests detecting disturbances in the blood retinal barrier

Fundus flourescein angiogram(FFA)

.The typical angiographic finding described by GASS and NORTON is the early dye leakage from perifoveal capillaries followed by progressive filling of cystic spaces at later phases. As the walls of the cyst don't stain, a petalloid pattern is produced in the angiogram. The central ends of the petals seem to correspond to cysts in the outer plexiform layer while the peripheral ends of petals correspond to cysts in the inner nuclear layer. Late staining in the optic nerve is due to leaking capillaries in optic nerve head.

Clinically, based on the flourescein leakage, five stage of PCME has been described.

Grade 0- no leakage

Grade 1- edema less than perifoveal area

Grade 2- mild perifoveal edema

Grade 3- moderate perifoveal leakage about 1 disc diameter

Grade 4-severe perifoveal leakage of more than 1 disc diameter

Side effects:

1. Extravasation and local tissue necrosis are the most serious complication which is very painful and may lead to subcutaneous nodule formation. This can be prevented by withdrawing blood into the syringe and scalp vein.
2. Nausea, vomiting and allergic reactions
3. Vasovagal shock
4. Anaphylaxis – hypotension, tachycardia, bronchospasm, itching
5. Tonic clonic seizures, thrombophlebitis, pyrexia

Characteristic feature of CME:

A. Petalloid pattern of staining of cysts in macula

B. Disc may leak or stain

C. Leak into the vitreous in late phase

B. Tests detecting retinal tissue thickening

1. Optical coherence tomography(OCT)

OCT is a noninvasive device that obtains cross sectional high resolution images of retina and thus may detect retinal thickening [26]. It was infrared light to detect relative change in reflection at optical interfaces by the method of low coherence interferometry. It may be thought of as being analogous to B scan USG, although it measures optical rather than acoustic reflection. It is possibly indicated in early detection and follow up of patients with macular edema. It is as effective as fluorescein angiography but superior in detecting axial distribution of fluid. It measures the echo time taken for light to reflect from different structures at varying disturbances analogous to B scan ultrasonography. OCT examination is indicated in early detection and follow up of patients with macular edema. It has been shown to have a high degree of reproducibility.

2. Retinal thickness analyser(RTA)

It is a rapid screening process which generates a detailed map of retinal thickening [27]. The advantage of RTA is to scan a relatively wide area in a short time.

3. Scanning laser ophthalmoscopy(SLO)

It can quantify retinal thickness by ophthalmoscopy and retinal topography [28]. It has been used to map retinal surface height revealing relative changes in the retinal surface height but not actual thickness. The axial resolution of SLO has been estimated at 300 micrometer whereas the RTA has a claimed depth resolution of 50micrometer. The SLO measures changes at various depths and converts these into changes in 2 planes ie depth of retina whereas RTA measures the distance between 2 peak reflection on an angled laser slit into a measure of retinal thickness. It is a rapid and noninvasive imaging method that provides quantitative analysis of macular cysts in addition to qualitative information. Advantages of SLO is scanning a small focused spot to generate an image , ability to image through small pupils, retinal hyperpigmentation, blood , heavy exudation or subretinal fluid. It has been used to assess the photoreceptor function in various stages of macular edema [29].

C. Tests assessing retinal function

Macular edema may potentially affect the macular function as far as visual acuity and contrast sensitivity are concerned. Tests assessing macular functions may be used indirectly to detect the effects of macular edema and follow up its treatment. Contrast sensitivity and ERG are both clinical and experimental tools.

1. Contrast sensitivity

Ibanez et al in a prospective comparative study evaluated the effect of PCME on contrast sensitivity. They reported a statistically significant decrease in contrast sensitivity for patients who developed transient or persistent PCME for all spatial frequencies studied at 2 months and for higher frequencies at 8.5 months following surgery as opposed to no CME group. Reduction in contrast sensitivity may account for the persistent difficulties experienced by patients despite good snellen visual acuity [30].

2. Electroretinogram(ERG)

Regardless of the visual acuity, eyes with PCME show reduction in amplitude of the oscillatory potentials[12] .This is said to be the most sensitive indicator of PCME. Oscillatory potentials are generated in the amacrine cells or inner plexiform layer and affected by any abnormality

of the retinal vasculature. Analysis of each component of foveal ERG can be used to quantify the severity of PCME [12].

TYPE 1: indicated by reduced amplitude of the oscillatory potential with normal a and b waves.

TYPE 2: characterized by reduced amplitude of oscillatory potential and b wave.

TYPE 3: comprises reduced oscillatory potential and a & b waves.

The mean time between cataract surgery and ERG changes was significantly longer for types 2 & 3 than for type 1.

A very important development in the ERG-field in recent years is the multifocal-ERG recording system. This system allows assessment of ERG activity in small areas of retinal dysfunction and allows the derivation simultaneously of 61 or 102 local ERG signals in a central visual field of about 60° diameter around the fovea in a considerably short time of 4 to 8 minutes. So the decrease or retinal function due to regional disorders in the outer retinal layers can be described in details by this technique, which allows the functional mapping of the retina.

MANAGEMENT OF CME

Management of CME includes the preventive measures to be taken and treatment which includes medical and surgical mode.

Preventive measures:

Although it is impossible at present to completely avoid PCME in all cases, a few precautions may prove important in reducing incidence and severity of this important postoperative complication.

- Bag fixation of IOL
- Use of PMMA IOL
- Avoidance of primary posterior capsulotomy
- Minimize microscopic light exposure
- Minimize operation time
- Use of UV filters and UV IOL
- Prophylactic steroids in high risk factors

1. **Adequate suppression of preoperative uveitis**

Uveitis if present is treated by periocular and systemic steroids & a quiet period preferably for 3 – 6 months prior to surgery would be ideal.

2. **IOL surgery**

Attention to finer details namely proper positioning of the IOL, avoidance of iris tuck and pupillary distortion helps in minimizing chances of PCME. In the bag placement of IOL is preferable. Operative manipulation is also kept to a minimum.

3. **Type of IOL**

PMMA IOL's instead of 3 piece IOL's with polypropylene haptics should be used.

4. **Posterior capsulotomy**

It should be avoided whenever possible. Even if required it should be delayed for upto 1 year postoperatively.

5. **Adequate management of posterior capsular rent**

Especially if associated with vitreous loss. This includes complete clearance of vitreous from the anterior chamber.

6. **Adjustments in operating microscope**

Use of ultraviolet filters to reduce the phototoxic effect of illuminating system in the operating microscope.

7. **Adjustments during surgery**

Several methods have been introduced to minimize the amount of light entering eye during surgery. Cornea is covered with a semiopaque material or filter or cellulose sponge. To block the light entering pupil during suturing and to minimize phototoxicity, insertion of IOL under air help to defocus the light. An air bubble may be placed in anterior chamber after IOL insertion as well as rotation of the eye 10 degree inferiorly by a superior rectus still helps to keep the illuminated image away from the fovea.

8. **Minimize operating time**

Prolonged operating time besides exposing eye for a greater length of time to extraneous forces like instruments and infusion fluids, also increase the total dosage of phototoxic elements. Thus minimizing the operating time reduces the

risk of prolonged blood aqueous barrier breakdown and photic inflammation which may predispose to CME.

9. Use of UV absorbing IOL

These have been used to prevent postoperative photic retinal damage and possibly CME. Komatsu et al [31] reported no statistically significant difference in visual acuity or incidence of CME on the basis of FFA performed 6 months postoperatively in patients with or without UV absorbing IOL's. Kraff [19] reported a significant decrease in early postoperative angiographic CME with the use of UV filtering PCIOL's.

10. Use of prophylactic steroids & NSAIDS

Use of topical prostaglandin inhibitors may prevent development of PCME [21]. This is particularly useful in prone eyes such as in diabetics, those with uveitis in fellow eyes when the eye has had CME.

TREATMENT

Various models of treatment have been tried for CME. These include drugs like NSAIDS both topical and systemic, corticosteroids including oral, periocular and intravitreal, immunosuppressive, oral carbonic anhydrase inhibitors and vitrectomy, hyperbaric oxygen therapy and grid LASER photocoagulation. The challenge concerning the management of macular edema arises in chronic and persistent cases for which a stepwise approach is optimal.

MEDICAL MANAGEMENT

1. Topical NSAIDS and topical steroids [32,33]

Medical therapy for established PCME has focused primarily on use of anti-inflammatory drugs. Therapeutic blockage of prostaglandin and other chemical inflammatory mediators is the mainstay of management in CME. Topical NSAIDS are useful as they inhibit cyclooxegenase enzyme which is required for the production of prostaglandins. Topical NSAIDS including ketoralac tromethanine 0.5%, diclofenac 1%, newer drugs like bromfenac and nepafenac 0.1% have been used either for the treatment of macular edema after cataract surgery [32,33,34,35] or prophylactically to prevent angiographic CME. Weiz et al [36], suggested that the topical ketoralac eyedrops can be used with satisfactory results for the treatment of chronic PCME identified more than 24 months after cataract surgery.

Topical corticosteroids also inhibit production of prostaglandins by inhibiting the enzyme phospolipase A2 [37]. They penetrate the corneal epithelium and reach anterior chamber and are potentially helpful in treating CME caused by chronic iridocyclitis. More recently it has been reported that treatment of acute usually significant PCME with topical ketorolac & prednisolone combination therapy appears to offer benefits over monotherapy with either agent alone as their synergistic activity result in rapid resolution of symptomatic CME [38].

Side effects of NSAIDS include ocular irritation conjunctival irritation, punctuate keratopathy and mydriasis. Recently reports of corneal melting following administration of topical diclofenac have been established [39,40].

Side effects of topical steroids include glaucoma, posterior subcapsular cataract, exacerbations of infection & recurrence of herpetic keratitis [41].

2. Corticosteroids

a. Periocular

The second step in the management of PCME is periocular steroids. Mc Cartney et al [42] used autoradiography to study penetration of hydrocortisone into both normal and inflamed eyes and found that the penetration was much faster in inflamed eyes. Injection can be given either as an orbital floor injection or as posterior subtenon injection.

Posterior subtenon injection according to theory is more likely to be effective due to closer location of the drug to macula. Freeman et al [43], looked at the location of repository steroid injection using A and B mode ultrasonogram immediately before and after subtenon injection was given. They found that in 57% of the injections given superotemporally and in 30% given infra temporally, there had been successful delivery of the drug to the macular region.

Side effects: Inadverent globe penetration, elevation of IOP, central retinal artery occlusion and cataract formation [44,45].

b. Oral / systemic corticosteroids

Very useful in treating inflammatory CME especially in bilateral or resistant cases.

Side effects: Peptic ulceration, osteoporosis, exacerbation of diabetes mellitus , hypertension, cushingoid state and adrenal suppression.

OTHER MODALITIES OF TREATMENT:

1. Carbonic anhydrase inhibitors

These drugs act by increasing fluid absorption across the retinal pigment epithelium [46,47]. They may alter the polarity of ionic transport systems in the retinal pigment epithelium through inhibition of carbonic andhydrase and gamma glutamyl transferase enzymes which inturn results in increased fluid transport across the pigment epithelium from the sub retinal space to choroid with the reduction of edema. Farber et al [49] studied 30 patients with CME secondary to chronic iridocyclitis and reported statistically significant visual improvement in group treated with acetazolamide. Response to treatment was better in younger than older individuals. Topical dorzolamide has also been used in the treatment of CME.

Grover et al [50] in the double masked cross over study reported that oral acetazolamide administered in 5 patients with retinitis pigmentosa was more effective than dorzolamide in managing chronic macular edema and improving the visual acuity and allows oxygen to diffuse from choroid to the inner retina , where it raises the oxygen tension and relieves hypoxia. Since no clinical trial has been done, there is no information available concerning its efficacy or safety.

3. Hyperbaric oxygen

It is suggested that constriction of perifoveal capillaries by hyperbaric oxygen may facilitate the reformation of damaged functional complexes in the capillary wall resulting in decrease in macular edema. Oxygen may be beneficial in aphakic and pseudophakic macular edema or chronic CME attributable to uveitis [51].

SURGICAL TREATMENT

A. Intravitreal triamcinalone

. Data in literature suggests that intravitreal triamcinolone acetonide is a promising therapeutic method for chronic pseudophakic CME resistant to medical treatment [53].

Side effects - glaucoma, cataract, deposition of cortisone crystals in the macular region, transient central retinal artery occlusion, rhegmatogenous retinal detachment, endophthalmitis.

B. Nd YAG LASER vitreolysis

Lysis of vitreous strands to the cataract wound with Nd YAG LASER may have a role in treating CME after cataract surgery in a selective group of patients[54]. However many patients have extensive iridovitreal and iridocapsular adhesions

that is not amenable to LASER vitreolysis. In addition risk of this treatment such as intraocular pressure elevation and retinal detachment should be considered carefully against the benefits.

C. Grid LASER photocoagulation

It was described as a method of treating CME using ruby and argon LASER photocoagulation[56]. Laser lesion in experimental animals shows a temporary breakdown of blood retinal barrier and a subsequent repair, as the retinal pigment epithelial cells adjacent to burns proliferate and slide to replace the necrotic cell. The new retinal pigment epithelial cells produce tight junctions in several weeks which restores the integrity of retinal pigment epithelial barrier[56]. An alternative hypothesis states that grid LASER by destroying photo receptors reduces the oxygen consumption of the outer retina and allows oxygen to diffuse from choroid to the inner retina where it raises oxygen tension and relieves hypoxia.

D. Intra ocular lens removal

Some patients with IOL related CME (patients with iris clip IOL's, rigid closed loop ACIOL, iris tuck by an IOL haptic or capture of the iris by lens) may benefit from removal or exchange of IOL [57].

E. Pars plana vitrectomy

Mild cases of CME resolve spontaneously and many persistent cases respond to medical management. However in a minority of eyes clinical CME persists despite aggressive medical treatment. This is likely to occur in eyes with vitreous adhesions to the cataract wound and other anterior segment structures [58,59].

Effect of vitrectomy in improving visual acuity in aphakic and pseudophakic eyes with chronic CME associated with vitreous incarceration to the cataract wound, adhesions to anterior segment structures or iris capture with posterior synechiae has been confirmed by several studies [60,61,62]. Patients undergoing a complete pars plana vitrectomy have greater visual improvement than those undergoing a limbal vitrectomy [61].

The surgical procedure includes pars plana vitrectomy with complete removal of vitreous strands to the cataract wound and other anterior segment structures, lysis of irido vitreal and irido capsular adhesions. If no improvement occurs after several months of aggressive medical treatment, vitrectomy should be considered [60].

Peymen et al [59], reported pars plana vitrectomy with internal limiting membrane peeling and intra vitreal triamcinolone acetonide in two patients with chronic pseudophakic CME resulted in anatomical, angiographic and functional

improvement. Additionally removing internal limiting membrane may allow better diffusion of intravitreally administered steroid to the macula. Vitrectomy is also beneficial in management of retained lens fragments[63].

COMPLICATIONS OF CME

1. Development of permanent retinal damage due to prolonged edema.
2. Spontaneous rupture of the inner wall of a large central cystoid space to form a lamellar hole. Retinal pigment epithelium in the base of the hole is undisturbed.
3. Cellophane maculopathy and macular pucker caused by epiretinal membrane may form either at the onset of CME or as a late complication of CME. When they occur it is less likely that visual acuity will return to normal even after the resolution of CME. Additionally, these membrane may peel spontaneously from the surface of the retina and good visual acuity is restored.
4. Prolonged CME may occasionally produce atrophy of the outer retinal layers. Macula may appear normal except for absence of foveal reflex.

REVIEW OF LITERATURE

Although cystoid macular edema following cataract surgery is recognized as most common cause of decreased vision in post op period, reported incidence of this post -op complication continues to be quite variable.

1. John R Wittpenn & Steven Silverstein [65] **(AMJ 2008 oct 146 issue 4)** did a randomized masked comparison of topical ketorolac 0.5% with steroid 1% vs steroid 1% eyedrops alone in very low risk cataract surgery patients for CME. They concluded that adding perioperative ketorolac 0.5% to postoperative steroids significantly decreases the incidence of CME in patients without any known risk factors for CME.
2. Keith A Walter & Amy J Estes [66] did a study on management of ocular inflammation following routine cataract surgery(**US Opthalmic review 2011 ;4-2 :97-100**). The purpose was to determine whether bromfenac as a single agent is just as safe undergoing cataract surgery was done. 200 eyes were analysed in each group.1st group received prednisolone 1% eyedrops 4 times/day for 2 weeks & then tapered over 3 weeks.2nd group received bromfenac 0.09% once daily 2weeks before surgery &4 weeks after surgery. They concluded that bromfenac is safe & as effective as topical steroid in

controlling postoperative inflammation even when used alone without increasing intraocular pressure.

3. CME in aphakic and pseudophakic eyes (**AMJ ophthalmol.1979 july 88(1) : 45-8**) was reported by Miami study group[67]. A prospective fluorescein angiography comparing the incidence of CME after intracapsular cataract extraction with implant, extracapsular cataract extraction with implant and intracapsular cataract extraction without implant was done. ECCE with implant and intact posterior capsule had a significantly lower incidence of CME than ICCE with implant surgery in 16 – 24 months post operatively.
4. David L. Epstein [68] (**AMJ ophthalmol.1977 Apr 83 (4) 501-3**) reported incidence of CME 13 years after cataract surgery in a patient. Fluorescein angiography demonstrated CME except for vitreous adherence to the inner surface of cataract wound. No other cause was made out.
5. Richard M. klein & Lawrence Yannuzzi[69] reported the incidence of CME in first week after cataract extraction. (**AMJ Ophthalmol 1976 May 81(5) : 614-5**). They studied 100 consecutive cases of cataract extraction. 75 of hundred eyes demonstrated CME. They conclude that incidence of CME in 1 st week after cataract surgery was low when compared to the reported incidence 4 – 6 weeks post operatively.

6. Sanfold L. Severin reported the incidence of late onset CME in pseudophakia [70] (**AMJ Ophthalmol 1980 Aug ;90(2) : 223-5**) after 1 year or more of cataract extraction. Out of 100 patients 3 patients developed CME, of which 1 patient resolved spontaneously and 2 patients did not resolve inspite of medical therapy.
7. Rapid response of CME following Nd YAG LASER capsulotomy to 0.5% ketorolac tromethamine solution was reported by Michael S Lee & Jonanthan H Lass[71] (**Ophthalmic surgery lasers imaging 2004 Mar – April 35(2) 162-4**) case report of 68 year old patient with CME following Nd YAG capsulotomy done for PCO was studied. Treatment with 0.5 % ketorolac eye drops had resulted in increase in visual acuity after 8 days of treatment and resolution of CME confirmed by FFA.
8. Flash, Allan J et al.[72] studied the effectiveness of ketorolac tromethamine 0.5% ophthalmic solution for chronic aphakic and pseudophakic CME(**AMJ ophthalmol 1987 apr 15 ;103(4) 479-86**).It was a double masked placebo controlled , randomized study which showed that the patients on ketorolac 0.5% eye drops in treatment of aphakic and pseudophakic macular edema had statistically proven significant improvement in visual acuity than those patients with placebo.

9. Post cataract CME by Manish Nagpal, Kamal Nagpal et al.[73], (**Ophthalmology Clinics Of North America 2001 Dec p651-657**) treatment with medical and surgical modes for CME was studied. Medical treatment includes topical periocular systemic steroids and topical NSAIDs and oral carbonic anhydrase inhibitors. Surgical treatment includes Nd YAG vitreolysis, Argon LASER photocoagulation, Pars Plana Vitrectomy.
10. David S Rh et al [74] – results of acute psuedophakic CME with diclofenac vs ketorolac was studied (**JCRS vol 29 (12) 2003 dec p 2378-84**).This was a randomized prospective study of 34 consecutive patients with clinical CME after uneventful phaco with PCIOL. Eyes with CME treated with 1 drop diclofenac 0.1% eyedrops 4 times per day or ketorolac eye drops 0.5%. Outcomes measured by observing improvement of CME and vision. Both methods resulted in significant decrease in CME and increased vision.
11. Chamblers William Stephens [75]studied the incidence of CME after phaco (**ophthalmology Vol 86(11) 1979 Nov p2019-2023**). 1055 consecutive cases of phaco were studied. Results showed decreased incidence of CME if posterior capsule is left intact permanently.
12. Heler, Jeffrey et al.[76], studied monotherapy with ketorolac eye drops and prednisolone eye drops versus combined therapy in treatment of acute pseudophakic CME (**ophthalmology vol 107 (11) 2000 Nov p2034-2038**).

28 patients after cataract surgery developed clinical CME in 21 to 90 days. They were given medical treatment. Results were combination therapy is better than monotherapy with ketorolac eye drops alone followed by prednisolone eye drops alone.

13. Allan J.Flach [77] reviewed the incidence, pathogenesis and treatment of cystoid macular edema following cataract surgery (**Trans American ophthalmology society-vol 96**; **1998,p 557-634**). He evaluated 7 laboratory and clinical studies to describe new information about the incidence, pathogenesis and treatment of CME following cataract surgery.
14. Iliff W Jackson [78]did a study on the phototoxic effect of relatively low intensity light on the retina and the suggestions by several authors that this might influence the development of CME in aphakic and psuedophakic patient [**aphakic CME and the operating microscope- is there a connection; Trans Am Ophthalmol soc.1985;83;476-500**). He suggested that operating microscope did not appear to be a significant factor in the development of CME but it represented a phototoxic threat to retina.
15. CME following ECCE with PCIOL implantation by Bradford J David; Wilkinson and Bradford Jr Reagan.H [79](**Retina – vol8; 1988- p 161-164**). A retrospective review of 20 psuedophakic eyes with PCIOL and clinical CME was performed to compare the outcome of this macular disorder to that

previously reported in both aphakic and psuedophakic eyes with iridocapsular or iris fixation IOL. Resolution of symptoms and apparent resolution of macular edema were observed in 18 cases. This study suggested that clinical CME occurring in association with PCIOL has a relatively fauvorable outcome. Inflammation produced by iris manipulation and by chronic iris trauma associated with iris supported IOL was the most important factor.

16. Jampol Lee M [80] (**Archives of ophthalmology,1988 vol 106 p 894-895**) did a prospective randomized clinical trial and concluded that there is increased incidence of CME in eyes that had undergone surgical capsulotomies than in those with eyes with intact posterior capsule.
17. Ursell Paul G et al [81] reported CME after phacoemulsification (**relationship to blood aqueous barrier damage and visual acuity JCRS vol 25 -11 1999 nov p 1492-1497**). In this study , out of 100 patients 19 case had angiographic CME postoperatively. This study showed that after phacoemulsification with CCC, the incidence of CME appears to be similar to that after ECCE.
18. Conway Mandi et al [82] did a non randomized retrospective study about Intravitreal triamcinolone acetonide for refractory chronic pseudophakic CME**(J cataract refractive surgery 2003 jan vol 29 p 27-33**). This was a

study of 8 eyes of 8 patients with a history of psuedophakic CME recalcitrant to current standard treatment modalities. Mean duration of CME was 20 months. Patients received intravitreal injections of 1mg triamcinolone acetonide and were followed for 8 mon. Visual acuity increased in all patients. Angiographic improvement occurred in all patients.

19. Jost B Jonas, Kreissig and Robert F [83] reported (**AJO 2003 vol 136 p 384-386**) reported the clinical outcome of intravitreal injection of triamcinolone acetonide as treatment of longstanding CME after phacoemulsification .The study included 5 patients who received intravitreal injection of 2.5 mg crystalline triamcinolone acetonide transconjunctivally with topical anaesthesia. All patients visual acuity improved but 2 patients developed increased IOP, which was controlled by antiglaucoma medications.
20. A direct correlation of the resolution of psuedophakic CME with acetazolamide therapy was reported by Tripathi , Ramesh C et al [84](**Annuals of ophthalmology vol 23-4 1991 april p 127-129**). Acetazolamide probably increases the rate of absorption of subretinal fluid by inhibiting carbonic anhydrase & gamma glutamyl transferase enzyme in the retina and retinal pigment epithelium.
21. Linda M Meyer & Carl Ludwig [85] (**Case report ophthalmol 2011;2 p 319-322**) presented a case report. A 83 year old patient suffering from CME

following cataract surgery in her left eye. She received 3 intravitreal inj of 0.4mg dexamethasone in 3 months following cataract surgery without any improvement in visual acuity. 7 months after cataract surgery, she received a single intravitreal injection of 0.7mg dexamethasone. 4 weeks later, her visual acuity improved and CME resolved with decrease in retinal thickness on OCT from 390 microns to 212 microns after OZURDEX (intravitreal dexamethasone implant).

AIMS AND OBJECTIVES

AIM:

1. To study the clinical profile of CME following cataract surgery.
2. To assess the response to various treatment modalities.

INCLUSION CRITERIA:

All cases of CME following cataract extraction.

EXCLUSION CRITERIA:

1. Any known case of preexisting cystoid macular edema secondary to causes other than cataract extraction like uveitis with cystoid macular edema, diabetic macular edema.
2. Prior intraocular surgery like trabeculectomy, retinal detachment and vitrectomy surgeries.
3. Drug induced cystoid macular edema like latanoprost.
4. Patients with any coexisting anterior segment or fundus pathology.

MATERIALS AND METHODOLOGY

The study had been conducted at ophthal department of Government Rajaji Hospital, Madurai between February 2012 to October 2012. All patients diagnosed as having CME after cataract surgery were included in this study. Diagnosis of CME was by clinically identifying cystoid macular edema in the macular area using slit lamp biomicroscopy with 78D or 90D lens. FFA or OCT was done in all cases at the time of diagnosis and at each follow up to correlate the course of CME before and after treatment.

INCLUSION CRITERIA:

All cases of CME following cataract extraction.

EXCLUSION CRITERIA:

1. Any known case of preexisting CME secondary to causes other than cataract extraction like uveitis with cystoid macular edema, diabetic macular edema.
2. Prior intraocular surgery like trabeculectomy, retinal detachment and vitrectomy surgeries.
3. Drug induced cystoid macular edema like latanoprost.
4. Patients with any coexisting anterior segment or fundus pathology.

METHODOLOGY:

1. OCULAR HISTORY

Patient age, presenting complaints, duration of symptoms and mode of onset was taken. Review of medical records was done to identify preop, intraop and postop risk factors.

2. SYSTEMIC HISTORY

Detailed history of diabetes mellitus and any other systemic illness was noted.

Detailed ocular examination including best corrected visual acuity, anterior segment and posterior segment examination by slit lamp biomicroscopy to intraop and postop risk factors for CME.

a. Visual acuity

BCVA was measured by subjective and objective refraction with Snellen chart .

b. SLE

SLE was carried out to look for

- Any iris or vitreous incarceration at the wound site
- Anterior chamber- cells and flare noted.

Number of cells were assessed and graded from 0 to +4 as follows (HOGAN'S GRADING)

<5	0
5-10	1+
11-20	2+
21-50	3+
>50	4+

Aqueous cells are graded according to number observed in an oblique slit beam 3mm long and 1mm wide with maximal light intensity and magnification

- Vitreous in anterior chamber
- Posterior capsular rupture
- PCIOL (in bag /sulcus)
- Cells in the anterior vitreous

Fundus illumination by slit lamp biomicroscopy with 90 lens:

All patients diagnosed as having CME were examined with slit lamp biomicroscopy using a noncontact lens after dilatation of pupil. Using thin angled slit beam, macula appears thickened with translucent intraretinal cystoid spaces which were best appreciated using red free light. Detailed fundus examination to rule out other causes of CME was also done.

c.FFA

Inspite of a thorough fundus examination, diagnosis of CME cannot be established without the use of fundus fluorescein angiography which remains the gold standard for diagnosis.

Detailed systemic examination were carried out for patients who underwent this procedure. Systemic side effects were explained to the patient and none of them experienced any serious adverse effect in our study. FFA was taken at the time of diagnosis and after treatment during subsequent follow up.

Procedure of FFA:

Procedure of FFA was informed to the patient. The patient was kept nil orally for atleast 4 hours prior to FFA. Pupil was dilated. Red free photographs were taken. Scalp vein was inserted and 3ml of 20% flourescein dye was injected. Colour photographs were taken. After the FFA, patient was reassured about the red stained urine and patient was observed for atleast 20 min.

c. OCT

OCT was done in patients wherever needed, at the time of diagnosis and after treatment during subsequent follow up. Macular thickness was measured at the time of diagnosis and after resolution of CME.

d. Intraocular pressure

IOP was recorded by goldmann applanation tonometer in patients who receive periocular and intravitreal steroids at the time of diagnosis of CME and during subsequent follow up.

TREATMENT GROUPS

Patients were considered into 3 groups depending upon the visual improvement as follows

GROUP A

Those patients whose visual acuity ranged from 6/6 – 6/18 were considered as group A. They were treated with topical steroids (prednisolone acetate 1% four times daily for 1 month) and topical NSAIDS (ketoralac tromethamine 0.5% four times daily for 1 month)& were followed up after 4 weeks. On follow up, IOP was measured in all patients and resolution of CME was noted. Those patients in whom CME did not resolve were treated as group B.

GROUP B

Those patients whose visual acuity ranged from 6/24 – 6/36 were considered as group B and they were treated with periocular steroid injections.

PROCEDURE OF POSTERIOR SUBTENON INJECTION

4% lignocaine eyedrops applied every 5 minutes for 3 times with cotton tipped applicator, superotemporal quadrant was anaesthetized. Upper eyelid was retracted and patient was instructed to look down and nasally. With tuberculin syringe 30mg of triamcinolone acetonide was given. These patients were followed up after 4 weeks and IOP was measured. Patients who had raised IOP were treated with topical antiglaucoma medications. Resolution of CME was noted and those in whom CME was not resolved, repeat injections were given.

GROUP C

Those patients whose visual acuity was <6/60 were considered as group C and treated with intravitreal steroids.

PROCEDURE OF INTRAVITREAL STEROIDS

This procedure was performed under sterile conditions in OT using an operating microscope and topical anaesthesia. Lids and adnexa were cleaned with povidone iodine. Lid speculum applied and 4mg of injection triamcinolone acetonide was withdrawn in 1ml tuberculin syringe and was injected transconjunctivally at a distance of 3-3.5 mm from the limbus. Eye was examined immediately after injection for the presence of central retinal artery pulsations and those with impending obstruction underwent paracentesis. All eyes were

reexamined at 15 minutes to half an hour after the injection to measure IOP and any immediate post injection complications. Patients were prescribed antibiotic eyedrops for atleast 3 weeks.

Patients were followed up after 2 weeks and IOP was measured and those patients whose IOP was high were treated with topical antiglaucoma drugs. They were followed up after 4 weeks and resolution of CME was noted.

TABLES AND GRAPHS

TABLE 1:

AGE DISTRIBUTION

Age in Years	No of patients	Frequency
<50	2	5%
51 – 60	18	45%
61 – 70	16	40%
>70	4	10%
Total	40	

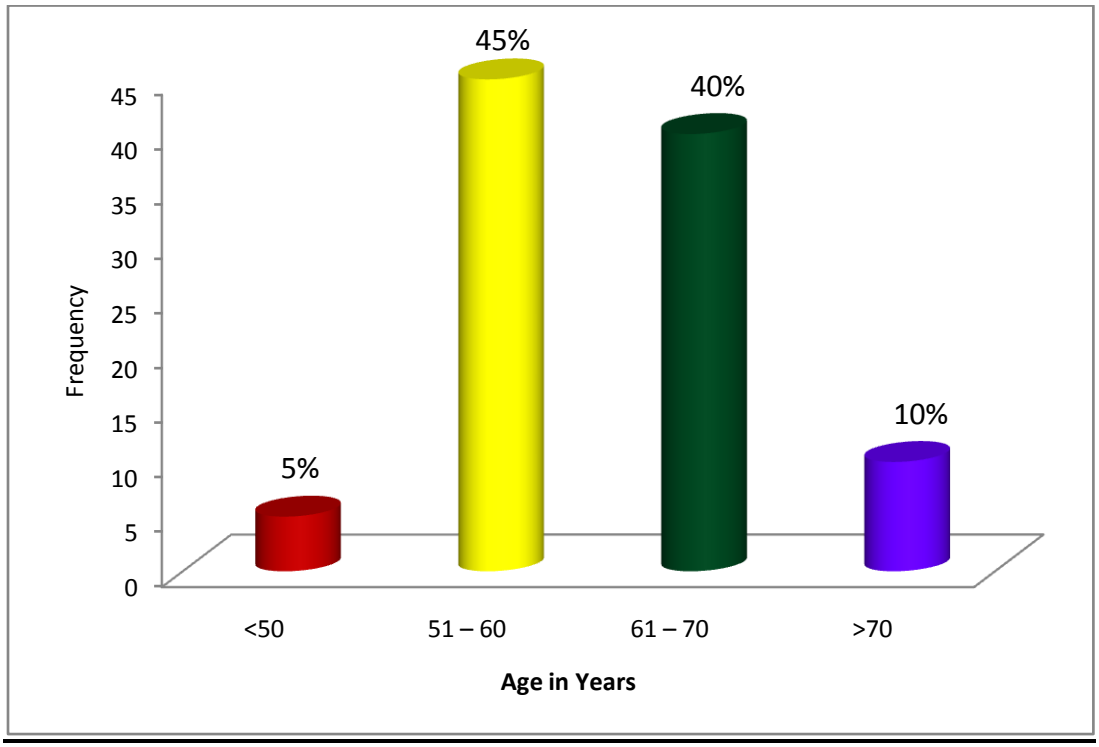


TABLE 2:

GENDER DISTRIBUTION

Gender	Frequency	Percentage
Male	26	65%
Female	14	35%
Total	40	

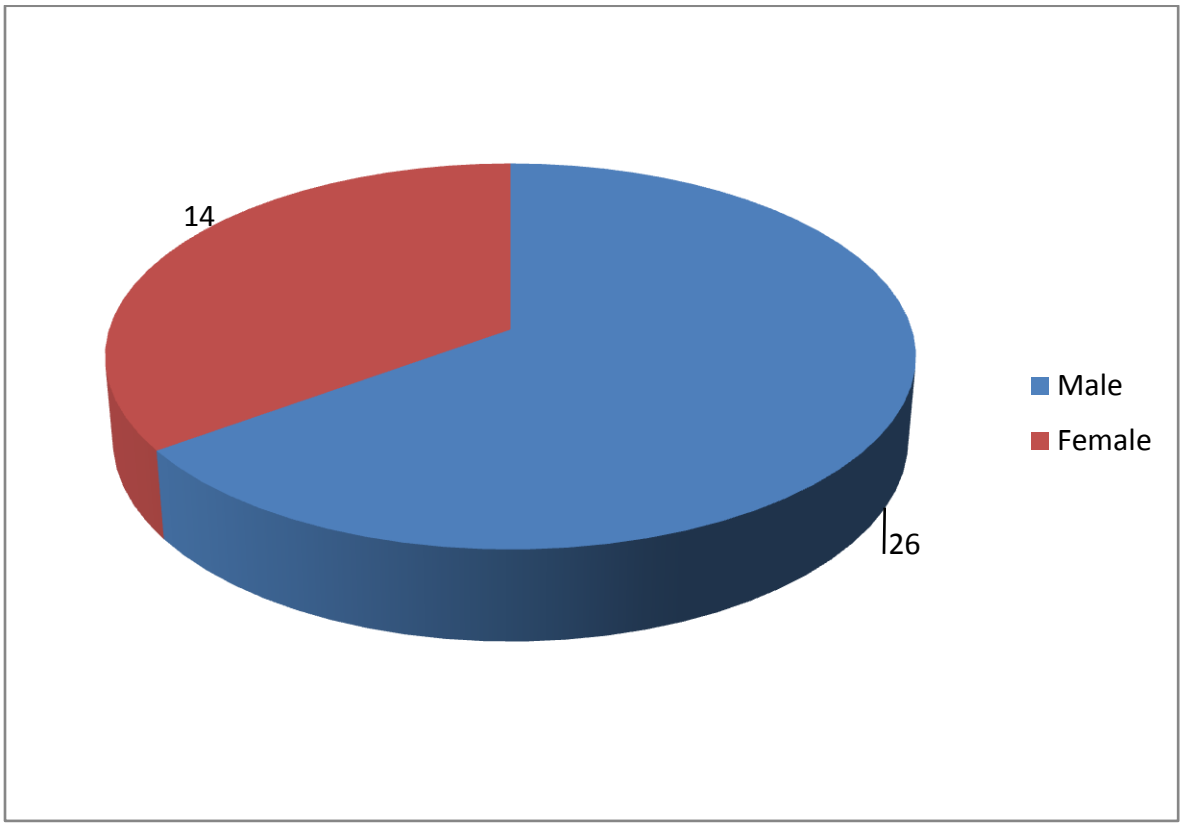


TABLE 3:

DURATION FROM CATARACT SURGERY

Duration	Frequency	Percentage
4 – 6 weeks	22	55%
7 -10 weeks	10	25%
11 – 24 weeks	6	15%
>24 weeks	2	5%
Total	40	

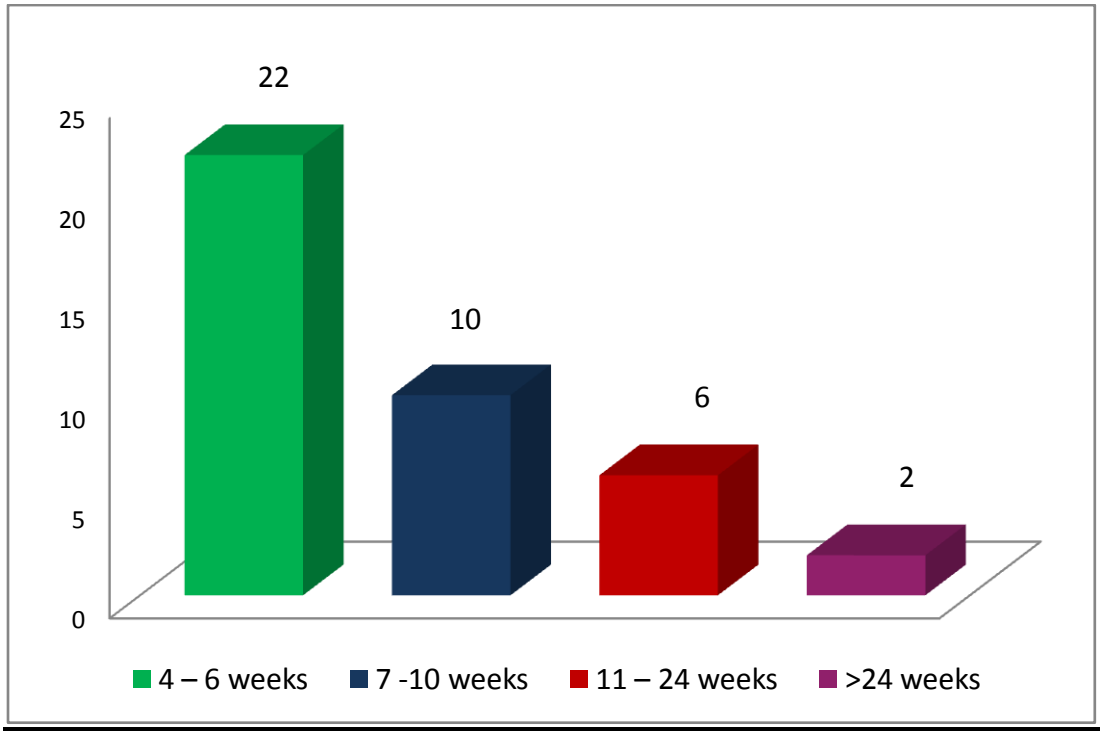


TABLE 4:

TYPE OF CATARACT SURGERY

Type	Frequency	Percentage
ECCE with IOL	18	45%
SICS with IOL	14	35%
PHACO with IOL	8	20%
Total	40	

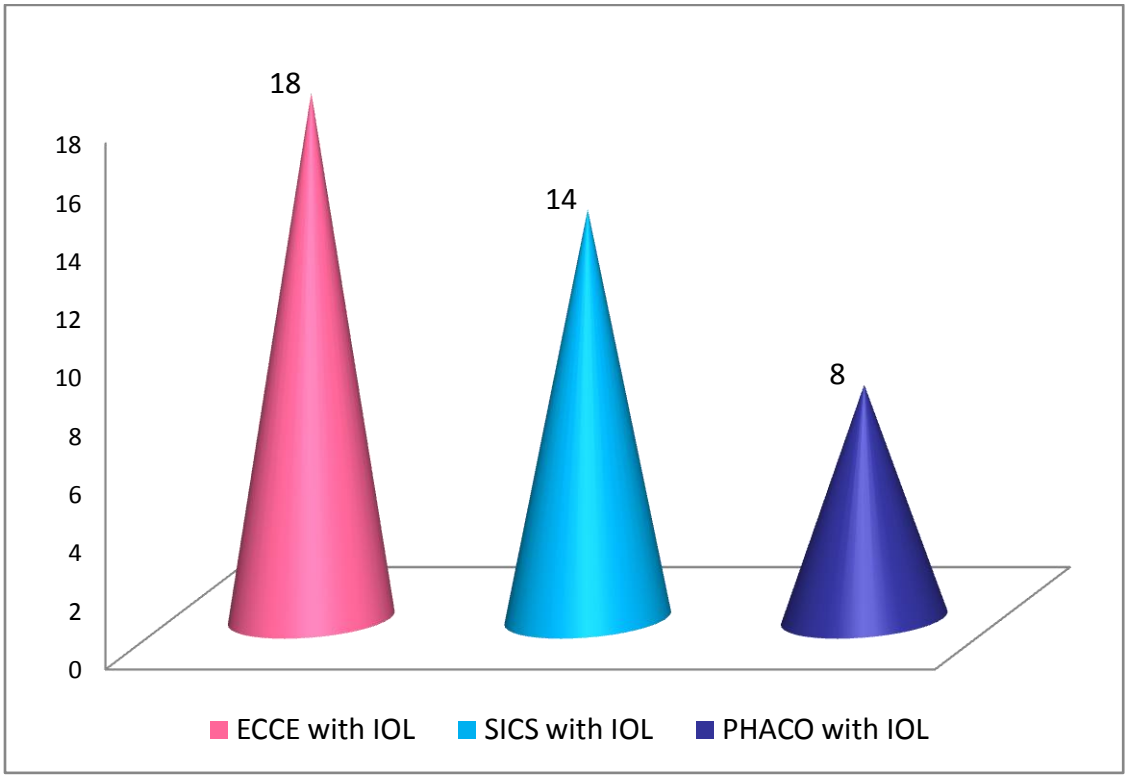


TABLE 5:

TYPE OF IOL

Type	Frequency	Percentage
PCIOL	38	95%
ACIOL	2	5%
Total	40	

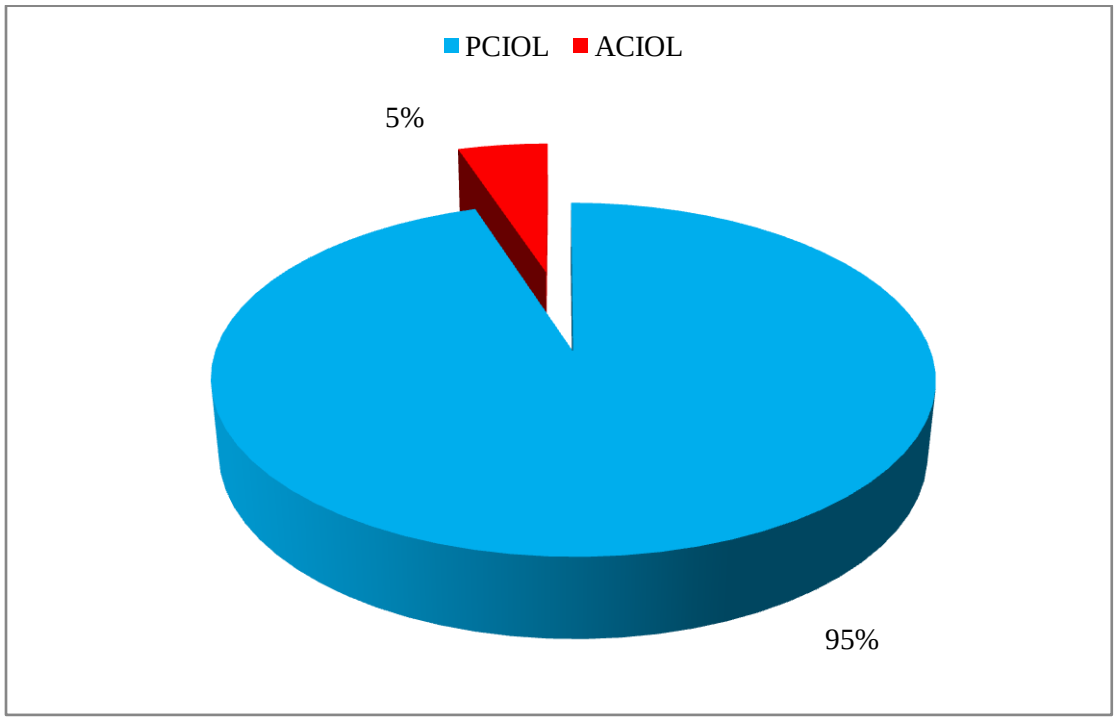


TABLE 6:

SYSTEMIC ILLNESS

Systemic illness	Frequency	Percentage
DM	4	10%
HT	4	10%
Both	2	5%
Nil	30	75%
Total	40	

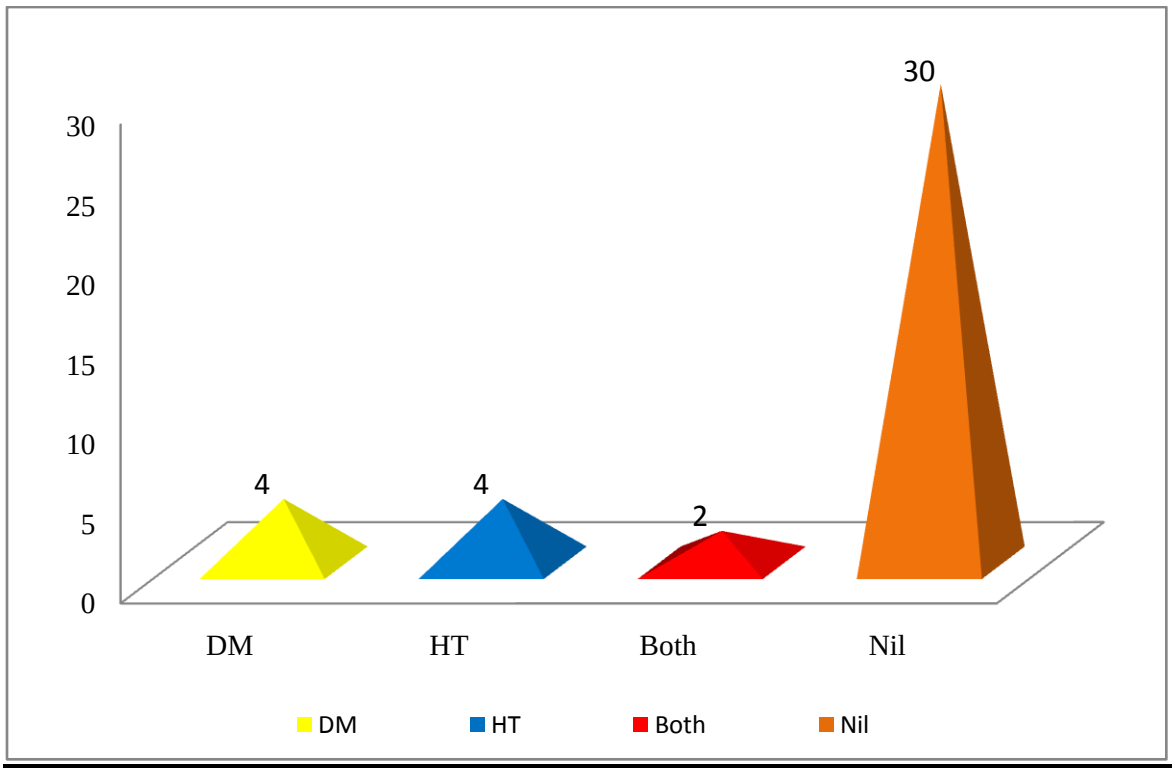


TABLE 6:

RISK FACTORS

Risk factors	Frequency	Percentage
Iris incarceration in Wound	2	5%
Vitreous in AC	2	5%
PCR	6	15%
Uveitis	18	45%
Vitritis	12	30%
Total	40	

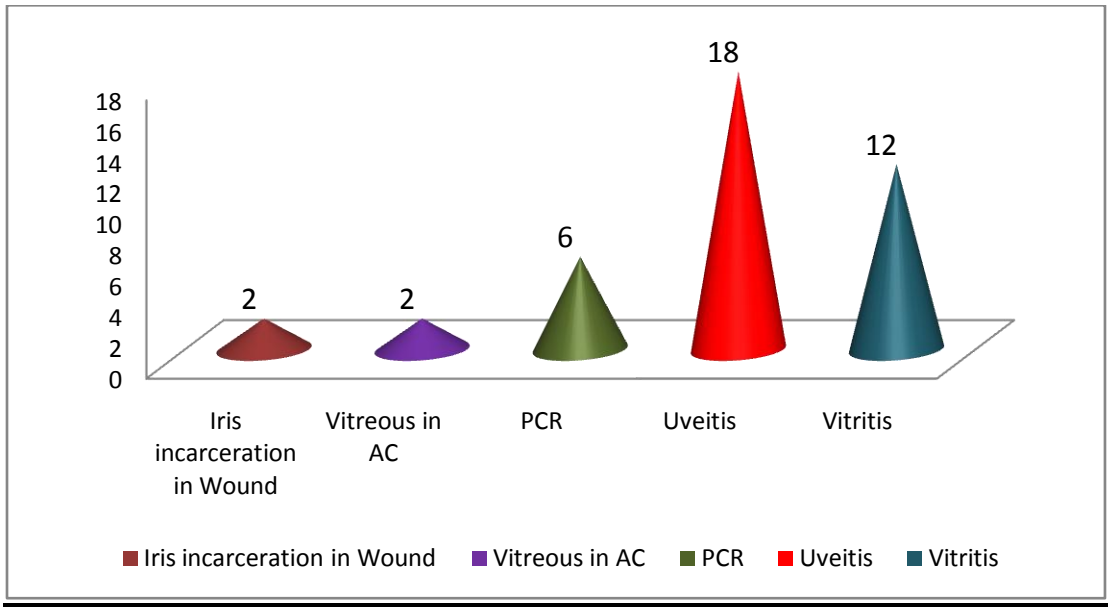


TABLE 8:

PRE- TREATMENT BCVA

Pre treatment BCVA	Frequency	Percentage
6/6 – 6/18	22	55%
6/24 – 6/36	12	30%
< 6/60	6	15%
Total	40	

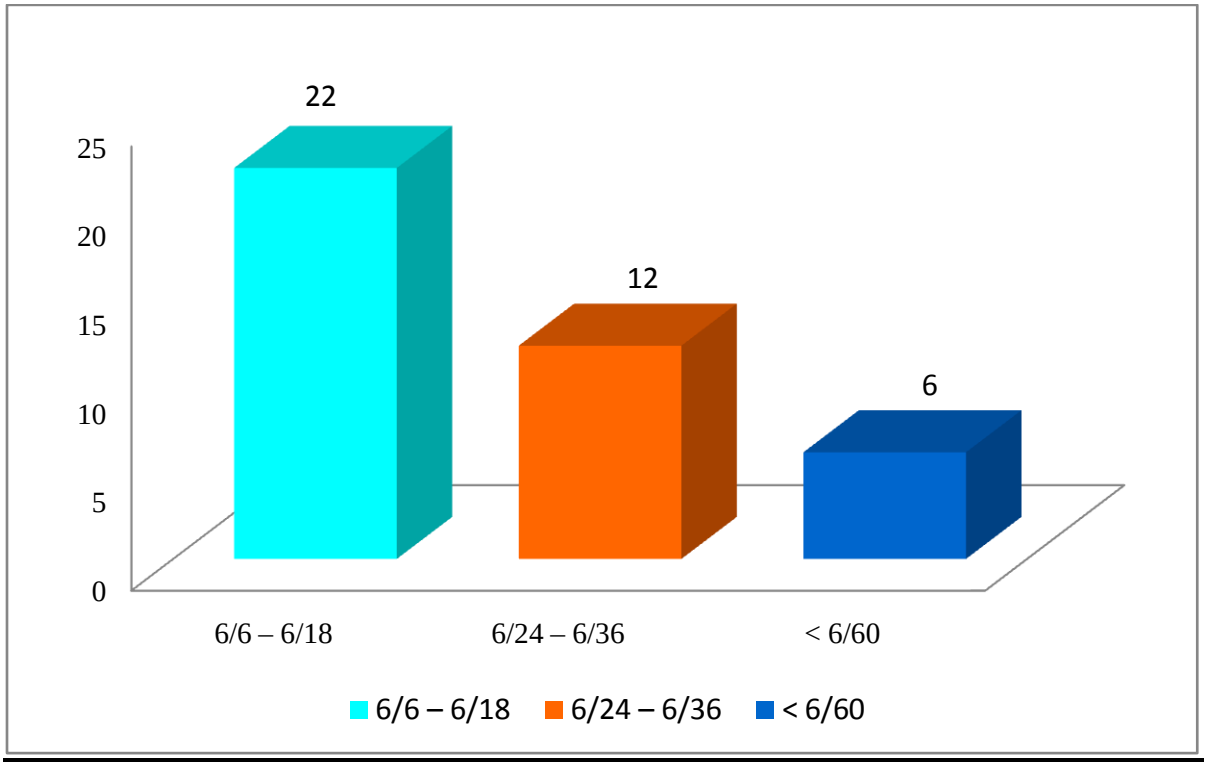


TABLE 9:

FINAL BCVA

Final BCVA	Frequency	Percentage
6/6 – 6/18	33	88.3%
6/24 – 6/36	5	8.4%
< 6/36	2	3.3%
Total	40	

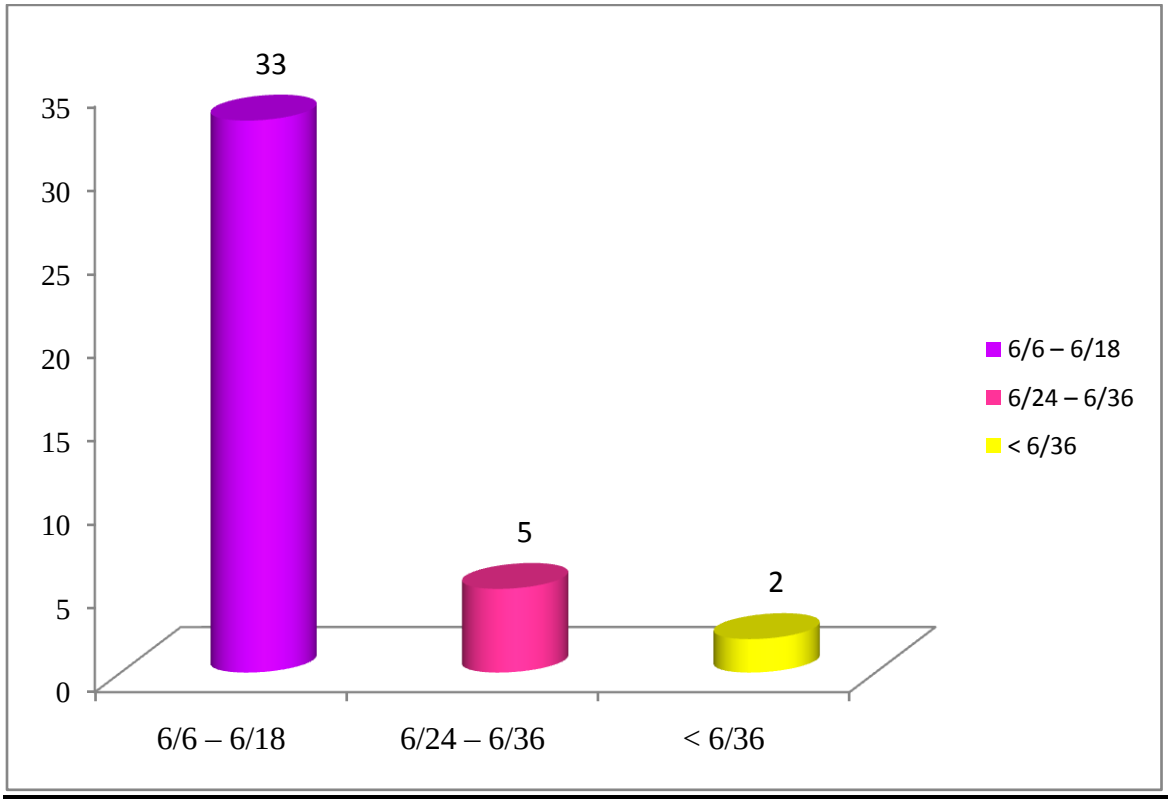


TABLE 10:

TREATMENT MODALITIES

Treatment modalities	Frequency	Percentage
Topical NSAIDS & Steroids	22	55%
Periocular steroids	12	30%
Intra vitreal steroids	6	15%
Total	40	

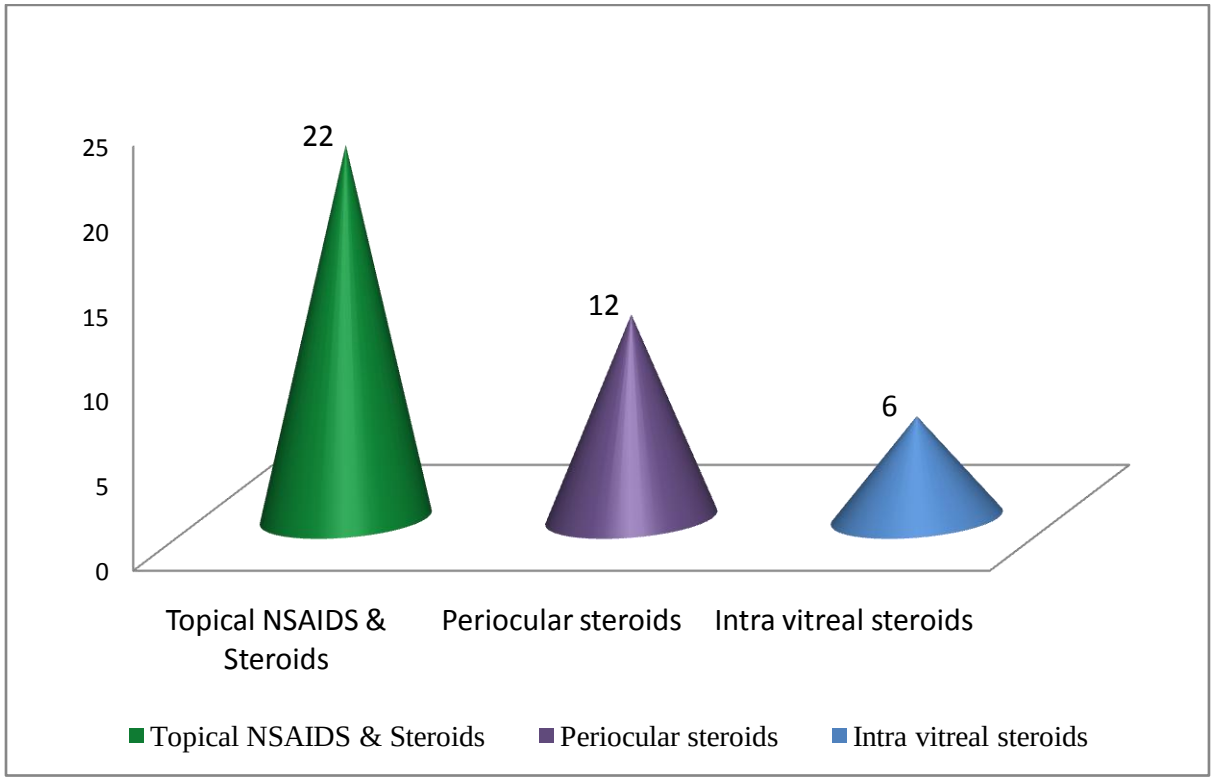


TABLE 11:

RESOLUTION OF CME

Resolution of CME	Frequency	Percentage
Resolved	35	87.5%
Not Resolved	5	12.5%
Total	40	

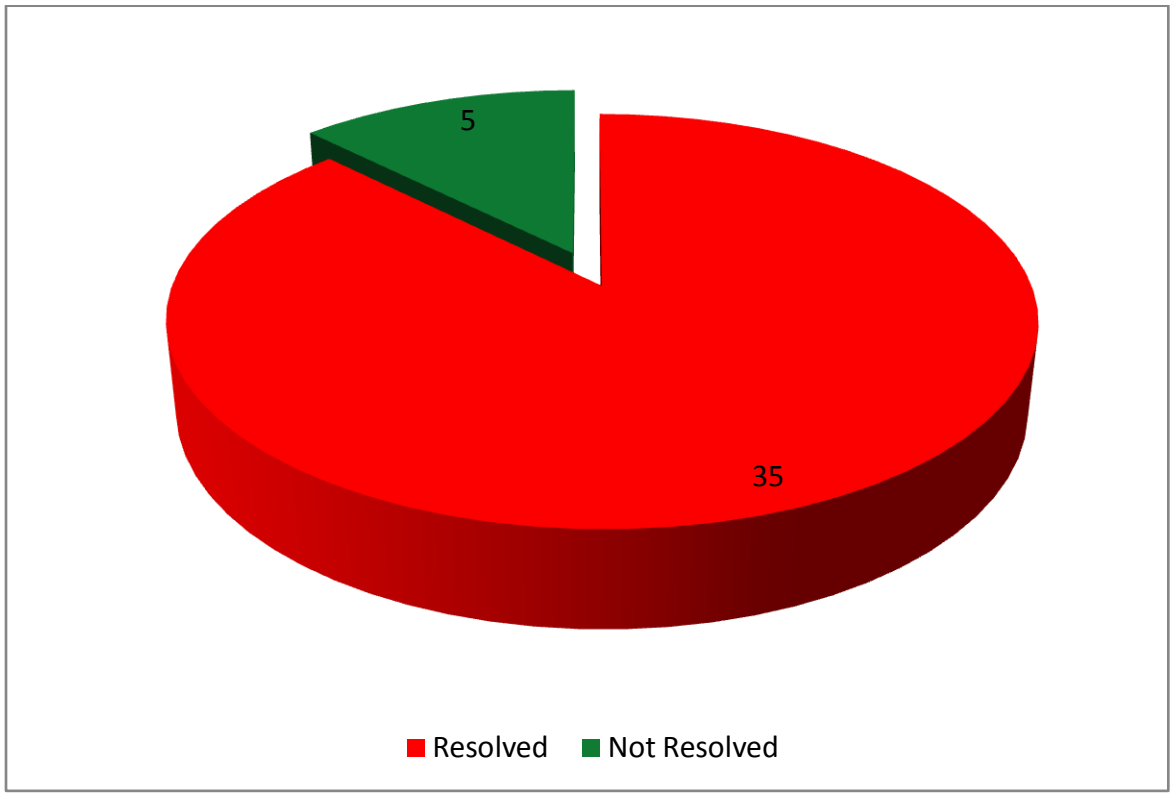


TABLE 12:

TOPICAL VS PERIOCULAR VS INTRAVITREAL STEROIDS FOR TREATMENT OF CME

Treatment given	Topical NSAIDS & Topical steroids	Periocular Steroids	Intravitreal Steroids
No of patients	22	12	6
Resolved	14 (64%)	9 (75%)	4 (67%)
Not Resolved	8 (36%)	3 (25%)	2 (33%)

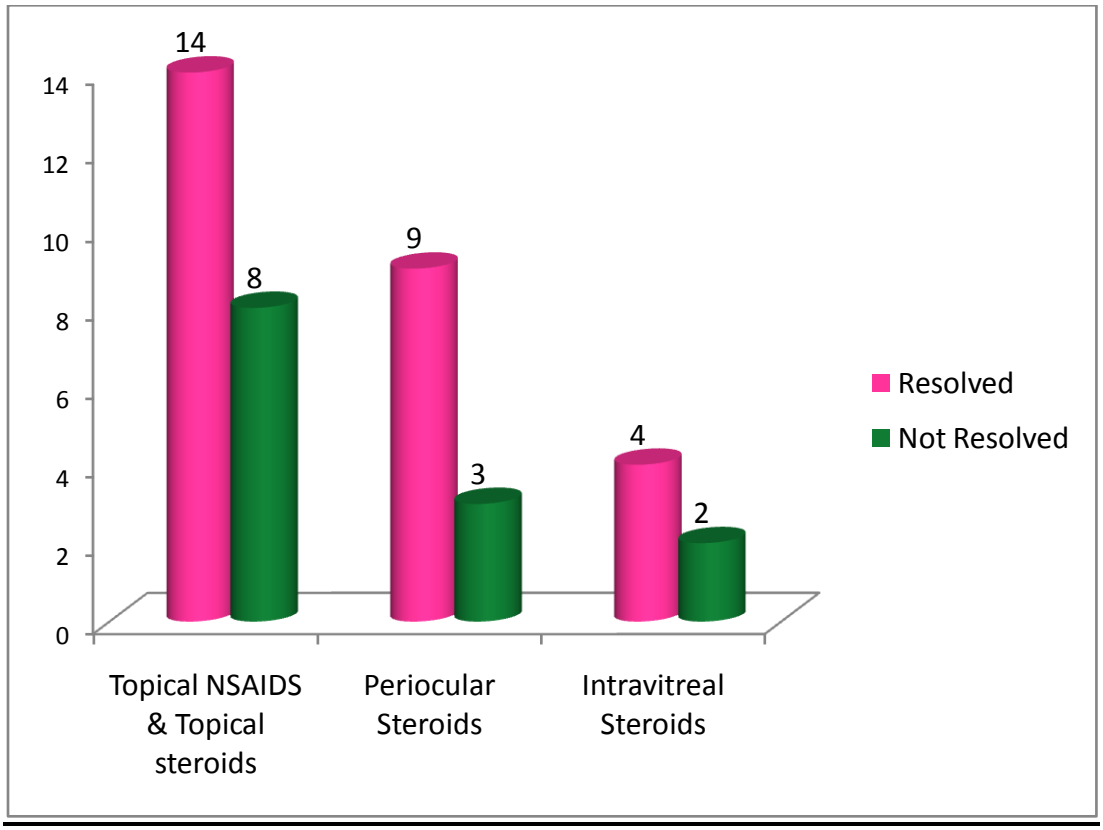
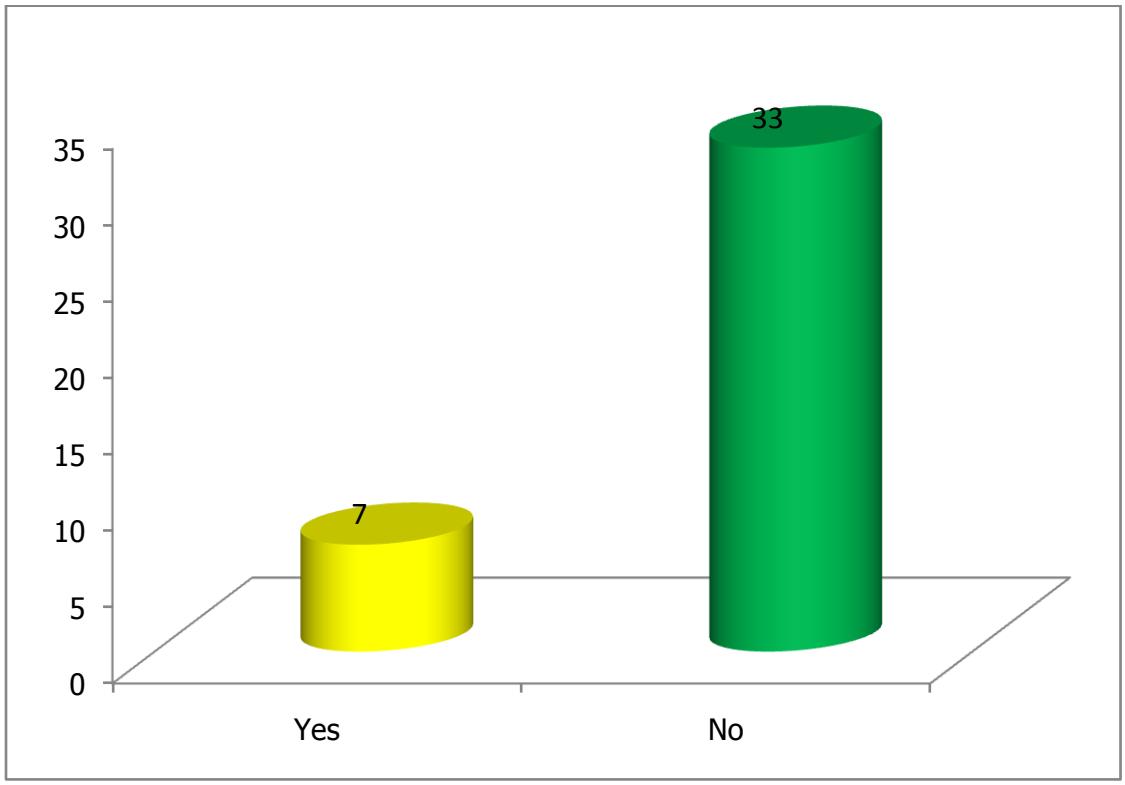


TABLE 13:

INCREASING IOP ON FOLLOW UP

Increasing IOP	Frequency	Percentage
Yes	7	17.5%
No	33	82.5%
Total	40	



DISCUSSION

CME after cataract surgery is the most common cause of decreased vision in the postoperative period. This clinical condition was first recognized by IRVINE in the year 1953 and now this condition is known as IRVINE GASS SYNDROME.

Most of the patients in our study developed CME in 4-6 weeks (55%). CME was most common in ECCE with IOL (45%) than SICS with IOL (35%) than PHACO with IOL (20%).

The most important intraoperative risk factor includes posterior capsular rupture associated with vitreous loss which cause breakdown in blood aqueous barrier and chronic inflammatory reaction. In our study, 6 patients had posterior capsular rupture as intraoperative complication in which 3 eyes with CME was not resolved due to formation epiretinal membrane, macular hole and macular degeneration.

In our study, major post operative risk factor include uveitis(18%) and vitritis (12%). These factors indicate that inflammatory mediators like prostaglandins and leukotrienes play a key role to cause post operative CME.

All patients were examined by slit lamp biomicroscopy with 90D lens and FFA was done in all patients at the time of initial diagnosis and subsequent follow up which showed resolution of CME after treatment.

As OCT is a noninvasive device, which obtains high resolution images of the retina and detects the presence of retinal thickening and it causes minimal discomfort to the patient, OCT was done in all cases at the time of diagnosis, at each follow up visit which showed resolution of CME and decrease in macular thickness after treatment.

Flanch Allan et al[72] had done a double masked placebo controlled randomized study in 1987 which indicated that ketorolac 0.5% eyedrops in aphakic and pseudophakic patients with macular edema showed that statistically significant visual acuity improvement than those patients with placebo.

As per Jeffrey et al study[76], combination therapy of ketorolac and prednisolone eyedrops in acute CME, who had done a randomized double masked prospective trial of 28 patients who developed clinical CME after 21-90 days postoperatively. Patients experienced recovery of 2 or more lines of visual acuity after combination therapy. They concluded that combination therapy appears to offer more benefit than monotherapy.

More recently, it has been reported that treatment of acute, visually significant PCME with topical ketorolac and prednisolone combination therapy appears to offer benefits over monotherapy with either drug alone as their synergestic effort results in rapid resolution of CME after treatment.

In our study, 22 patients presented with CME between 4-6 weeks postop period whose visual acuity range from 6/6- 6/18 was treated with administration of topical ketorolac tromethamine 0.5% eyedrops 4times per day for 1 month and topical steroids 1% eyedrops for 1 month. 14 patients showed resolution of CME at 1st follow up (i.e.) after 4 weeks. In remaining 8 patients CME was not resolved and they were treated with periocular steroids (i.e.) posterior subtenon injection of triamcinolone acetonide 30mg. CME was resolved in 8 cases and in 2 cases visual acuity did not improve due to macular degeneration.

2nd step in the treatment of postop CME is periocular steroids. According to Mccartney et al, injection triamcinolone acetonide 40mg given as posterior subtenon injection is more likely to be effective due to closer location of drug to the macula.

In our study, 12 patients who presented with CME between 4-6weeks postop period whose visual acuity range from 6/24-6/36 was treated with

posterior subtenon injection triamcinolone 30mg. Out of 12 patients, 9 of them showed resolution of CME in 1st follow up(i.e.) after 4 weeks. In 3 patients, CME was not resolved and they were given repeat triamcinolone 30mg by posterior subtenon route. At the final follow up also, these 3 patients did not show improvement in visual acuity due to formation of epiretinal membrane, macular hole and macular degeneration. And they showed increased IOP in the range of 24-28mm Hg and were treated with timolol 0.5%eyedrops twice daily.

3rd step in treatment of CME is intravitreal triamcinolone acetonide. Conway mandi et al[82] had done randomized retrospective study about intravitreal triamcinolone acetonide for refractory chronic PCME. A study of 8 eyes of 8 patients with history of PCME recalcitrant to current standard treatment modalities. Patients received intravitreal injection of 1mg triamcinolone acetonide and were followed up for 8 months. Visual acuity was increased in all patients and there were temporary increase in IOP which were easily controlled with topical antiglaucoma drugs.

In our study, 6 patients whose visual acuity was <6/60 were treated with injection intravitreal steroids (i.e.)intravitreal triamcinolone acetonide 4mg. They were reviewed after 2 weeks and complication like raised IOP was noted in 3 patients. And they were reviewed after 4 weeks and resolution of

CME was noted. Out of 6 patients, 4 of them showed resolution of CME and in 2 patients CME was not resolved due to chronic CME.

4th step in management of CME is pars plana vitrectomy. In our study, none of the patients underwent PPV. William Harton et al

reviewed 24 consecutive cases who underwent PPV in 1 eye for PCME. All 24 patients failed to improve on medical therapy and had preop evidence of either vitreous adhesion to anterior segment structures or iris capture with IOL. 17 patients experience 3 or 4 lines of postop visual acuity improvement and all 24 patients had atleast 1 line improvement.

In our study, out of 40 patients 5 patients CME was not resolved even at the final follow up. Of which 3 patients who had posterior capsule rupture associated with vitreous loss as an intraoperative complication and they had uveitis postoperatively and they were treated with periocular steroids at the time of diagnosis of CME and they were reviewed after 4 weeks, since CME was not resolved, the injection was repeated. At the final follow up also, CME was not resolved due to formation of epiretinal membrane, macular hole and macular degeneration. 2 patients who had zonular dialysis as an intraoperative complication and they had uveitis and vitritis postoperatively

and they were treated with intravitreal steroids. CME was not resolved due to chronic CME.

Out of 40 patients, 7 patients showed raised IOP after 4 weeks of treatment. Out of 7 patients, 2 of them who received posterior subtenon injection once and 2 patients who received posterior subtenon injection twice showed raised IOP in the range of 24-28mm Hg and 3 patients who received intravitreal steroids showed raised IOP in the range of 28-30mm Hg. All these patients were treated with topical 0.5% timolol eyedrops twice daily and their IOP were under control.

SUMMARY

- Our studies included 40 patients with CME following cataract surgery with IOL of which 38 patients (95%) were implanted with PCIOL and 2 patients (5%) with ACIOL. Out of 40 patients 26 (65%) were men and 14 (35%) were women.
- 22 patients (55%) developed CME in 1st 4-6 weeks postoperative period and 10 patients (25%) in 6-10weeks and 6 patients (15%) developed CME after 11-24 weeks and 2 patients (5%) developed CME after 24 weeks.
- 18 patients with CME underwent ECCE with IOL (45%), 14 underwent SICS with IOL (35%) and 8 patients underwent PHACO with IOL (20%).
- Among systemic diseases, 4 patients had Diabetes mellitus, 4 patients had Hypertension, 2 patients had both diabetes and hypertension.
- Among 40 patients, none of the patients were treated with preop topical NSAIDS for preop uveitis. 4 patients were found to have CME in fellow eye.
- Among 40 patients, 6 patients (15%) had posterior capsular rupture associated with vitreous loss intraoperatively and had PCIOL implantation after anterior vitrectomy.

- Among 40 patients, 2 patients had ACIOL implantation due to >180 degree zonular dialysis.
- Postoperative examination revealed 18 patients (45%) had uveitis, 12 patients (30%) had vitritis, 2 patients (5%) had iris incarceration in wound. 2 patients (5%) had vitreous in anterior chamber.

GROUP A:

In our study 22 patients whose visual acuity range from 6/6 -6/18 were considered as group A. Mean pretreatment visual acuity was 6/13. Mean pretreatment macular thickness was 340microns.

They were treated with topical steroids (prednisolone 1% eyedrops 4 times daily for 1 month) and topical NSAIDS (0.5% ketolorac eyedrops 4 times daily for 1 month) at initial diagnosis of CME.

These patients were reviewed after 4 weeks and their IOP was measured by Goldmann applanation tonometer. Out of 22 patients, 14 patients showed resolution of CME. Their mean post treatment visual acuity was 6/9.2. Their mean post treatment macular thickness was 250 microns.

In 8 patients, CME was not resolved with topical steroids and topical NSAIDS. They were treated with periocular steroids (triamcinolone acetonide

30mg by posterior subtenon route). These patients were reviewed after 4 weeks. CME was resolved in all 8 patients both clinically and by OCTwise.

6 patients showed visual acuity improvement by 1 to 2 lines and in 2 patients visual acuity remained the same. Macular degeneration is the cause for non improvement of visual acuity in these cases.

In one patient, IOP was high level (26mmHg) and patient was treated with 0.5% timolol maleate eyedrops two times per day.

GROUP B:

12 patients whose visual acuity ranged from 6/24 – 6/36 were considered as group B. Mean pretreatment visual acuity was 6/26 and pretreatment macular thickness was 480microns. They were treated with periocular steroids (posterior subtenon injection of triamcinolone acetonide 30mg). Before giving periocular steroids, IOP was measured.

These patients were reviewed after 4weeks. Out of 12 patients, 9 patients showed resolution of CME. Mean post treatment visual acuity was 6/30 and mean post treatment macular thickness was 300microns. IOP was measured in the 1st follow up. In 3 patients, CME was not resolved and they were treated with repeat posterior subtenon injection. At the final follow up also, these 3 patients did not show improvement in visual acuity due to epiretinal membrane, macular hole and

macular degeneration. They showed raised IOP in the range of 24-28mmHg and were treated with 0.5% timolol eyedrops two times per day.

GROUP C;

6 patients whose visual acuity was <6/60 were considered as group C. Their mean pretreatment visual acuity was 6/120 and pretreatment macular thickness was 780microns.

Since they presented with significant CME, they were treated with intravitreal steroids (triamcinolone acetonide 4mg). Before giving injection, IOP was measured.

They were reviewed after 2 weeks and complications like raised IOP noted and again reviewed after 4 weeks and resolution of CME was noted.

Out of 6 patients, 4 patients showed resolution of CME and in 2 patients CME was not resolved. Their mean post treatment visual acuity was 6/80 and mean post treatment macular thickness was 350microns. 2 patients visual acuity got worsened and the reason was due to chronic CME. 3 patients showed raised IOP in the range of 28-30mm Hg and were treated with timolol 0.5% eyedrops twice daily.

CONCLUSION

- In our study, CME was the most common vision threatening complication following cataract surgery who presented with defective vision in 1st 4-6 weeks postoperatively.
- Periocular steroids were found to be most effective form of treatment.
- Topical NSAIDS and topical steroids were also effective in treating Psuedophakic cystoid macular edema and intravitreal steroids can be given in resistant cases. But periocular and intravitreal steroids most commonly associated with raised IOP compared to topical steroids.
- Other modalities of treatment like pars plana vitrectomy and grid laser photocoagulation should be considered in resistant cases.

BIBLIOGRAPHY

1. Irvine SR: A newly defined vitreous syndrome following cataract surgery, AJO, vol. (36): may 1953; 599-619.
2. Gass JD, Nortan EW: CME and papilloedema following cataract extraction, Arch Ophthalmol., vol (76):1966; 646-61.
3. Severin SL: Late CME in psuedophakia, AJO, vol.(90-2):1980; 223-225.
4. Nagpal M, Nagpal K, Nagpal PN: Post cataract CME, Ophthalmology Clinics North America, vol (14): dec 2000; 651-659.
5. Cunha Vae JG,Travassos A: Breakdown of blood retinal barrier and cystoid macular edema, Surv. Ophthalmol, vol(28) suppl., 1984;485-92.
6. Scephens CL, Avila MP, Jalkh AE, Trempe CL: Role of vitreous in CME, Surv. Ophthalmol 28 suppl: 1984; 499-504.
7. Newson, William A, C Ian Hood and Jeffrey A Horwitz:

   CME- Histopathologic and angiographic correlations: Trans Am Acad Ophthalmol: vol.76(4): july-aug 1972;p.1005.
8. Fine BS, Bucker: Macular edema and cystoid macular edema, AJO, vol (92): 1981;466-481.
9. Wolter JR: Histopathology of CME, Graefer Arch Clini.Exp. Ophthalmic, 216:1981;85-101.

10. Ibanez HE, Lesher MP, Singerman LJ et al: Prospective evaluation of the effect of pseudophakic CME on contrast sensitivity, Arch Ophthalmol 11(2):1993: 1635-39.
11. Spaide RF, Yannuzi LA ,Sisco LJ: Chronic CME and predictors of visual acuity, Ophthalmic Surgery 24(4): 1993;262-67.
12. Miyake K: Flourophotometric evaluation of blood ocular barrier function following cataract surgery and intraocular lens implantation,JCRS, vol 14:1988;560-68.
13. Bergman M, Laatikainen L : CME after complicated cataract surgery and implantation of ACIOL, Acta ophthalmol ,72(2):1994;178-80.
14. Kraff MC,Sanders DR, Jampol LM et al: Effect of primary capsulotomy with cystoid macular edema, AMJ , 98(2):1984;166-170.
15. Guez Cosier, Othenin Girard P, Herbert CP: Differential treatment of postoperative uveitis induced inflammatory CME(French) , klin monatsbl augenkeika, 200(5): 1992; 367-73;
16. Byrnes GA, Antoszyk AN, Mazui PO et al: Photic maculopathy after ECCE, a prospective study, Ophthalmology 99(5): 1992; 731-37.
17. Kraff MC, Sanders DR, Jampol LM et al: Factors affecting psuedophakic CME- five randomized trials, J Am intraocular implant Soc., 2(4): 1985; 38-50.

18. Flack AJ, Stegman RC, Graham J et al: Prophylaxis of aphakic CME without steroids- a paired comparison, placebo controlled double blind study, Ophthalmology, 97(10): 1990; 1253-58.
19. Iwase K, Shimizu K: PCIOL implantation among diabetic patients- examination of CME and maculopathy, Nippon Ganka Gakkai Zasshi Acta Ophthalmologicae Japonicae, Tokyo 94(6): 1990; 586-92.
20. Gass JD, Anderson, Da Vis EB: A clinical fluorescein angiographic and electron microscopic correlation of CME, AJO, 100: 1985; 82-6.
21. Kylstra JA, Brown JC, Jaffe GJ et al: The importance of fluorescein angiography in planning laser treatment of diabetic macular edema, Ophthalmology, 106: 1999; 2068-73.
22. Mu MR, Puliafita CA, Duker JS et al: Topography of diabetic macular edema with optical coherence tomography, 105: 199; 360-70.
23. Zermer RC, Shahidi M, Mori MT, Benhamou E: In vivo evaluation of a non invasive method to measure retinal thickness in primates, Arch Ophthalmol.,107: 1989;1006-9.
24. Mainster MA, Timberlake GT, Webb RH, Hughes GW: Scanning laser ophthalmoscopy, clinical applications, ophthalmology, 89: 1982; 852-7.

25. Ibanez HE, Lesher MP, Singermann LJ et al: Prospective evaluation of the effect of pseudophakic CME on contrast sensitivity, Arch Ophthalmol., 11(2), 1993; 1635-39.

26. Komatsu M, Kanagami S, Shimizu K : Ultraviolet absorbing intraocular lens- comparison of angiographic CME, J Cataract Refract Surg., 15(6): 1989; 654-57.

27. Peterson M ,Yoshiumi MO, Helper R et al : Topical indomethacin in the treatment of chronic CME, Graefes Arch Clini. Exp. Ophthalmol., 230(5): 1992; 401-05.

28. Civerichia LL, Balent A: Treatment of pseudophakic CME by elevation of IOP, Ann Ophthalmol., 16(9): 1994; 890-94.

29. Burnett J, Tessler H, Isen Berg S, TSO MO: Double masked trial of fenoprofen sodium-treatment of chronic aphakic CME, Ophthalmic Surg., 14 : 1983; 150-2.

30. Miyake K : Indomethacin in treatment of postoperative CME, Surv. Ophthalmol., 28(suppl) 1984; 554-68.

31. Weisz JM, Bressler NM, Bressler SB, Schachat AP: Ketorolac treatment of psuedophakic CME identified more than 24 months after cataract surgery, Ophthalmology, 106: 1999; 1656-9.

32. Lin JC, Rapuano CJ, Laibson PR et al: Corneal melting associated with use of topical nonsteroidal anti-inflammatory drugs after ocular surgery, Arch. Ophthalmol., 118: 2000; 1129-32.

33. McGill J: The enigma of herpes stromal disease, Br.J. Ophthalmol., 71 : 1987; 118-25.

34. Freeman WR, Green RI, Smith RE: Echographic localization of steroids after periocular injection, AJO, 32: 1998; 385-91.

35. Nusseublatt RB: The natural history of uveitis, Int. Ophthalmol., 14, 1990; 303-8.

36. Yoshikawa K, Kotake S, Ichiisi A et al: Posterior subtenon injections of repository steroids in uveitis patients with CME,Jpn J Ophthalmol., 39: 1995; 71-6.

37. Marmor MF, Abdul Rakim AS, Cohen DS: Effect of metabolic inhibitors on retinal adhesions and subretinal fluid resorption, Invest Opthalmol Vis Sci., 19: 1980; 893-903.

38. Marmor MF, Maack T: Enhancement of retinal adhesion and subretinal fluid absorption of acetazlamide, Invest Opthalmol Vis Sci., 109: 920-7; 2002.

39. Kitu M. Marmor MF: Effects on retinal adhesive force invivo of metabolically active agents in the subretinal space, Invest Opthalmol Vis Sci., 33: 1992; 1883-7.

40. Farber MD, Lam S, Tessler HH et al: Reduction of macular edema by acetazolamide in patients with chronic iridocyclitis–a randomized prospective crossover study, Br. J. Ophthalmol., 78: 1994; 4-7.

41. Grover S, Fishman GA, Fiscella RG, Addman AE: Efficacy of dorzolamide hydrochloride in the management of chronic CME in patients with Retinitis pigmentosa, Retina, 17: 1997; 222-31.

42. Young S, Larkin G, Branley M, Lightman S: Safety and efficacy of intravitreal triamcinolone for CME in uveitis, Clinic. Exper. Ophthalmol., 29: 2001; 2-6.

43. Conway MD, Canakis, Livir Rallatos C, Peymem GA: Intravitreal triamcinolone acetonide for refractory chronic psuedophakic CME, J Cataract Refract Surg., 29: 2003; 27-33.

44. Katzen LE, Fleishman JA, Trokel S: Yag Laser treatment of CME, Am J Ophthalmol 95: 1983; 589-92.

45. Smith SG: Intraocular lens removal for chronic CME, J Cataract Refract Surg.,15: 1989; 442-5.

46. Evaluation of grid pattern photocoagulation for macular edema in central retinal vein occlusion, central retinal vein occlusion study group, ophthalmology 102: 1994; 1425-33.

47. Wilkinson CP, A long term follow up study of CME in aphakic and pseudophakic eyes: Trans Am Ophthalmol., soc. 79: 1981; 810-39.

48. Pendergast SD, Margherio RR, Williams GA, Cox MS: Vitrectomy for chronic psuedophakic CME, Am J Ophthalmol.,128: 1999; 317-23.

49. Peyman GA, Canakis C, Livir Rallatos C, Conway MD: Effect of internal limiting membrane peeling on chronic recalcitrant psuedophakic CME, A Report of 2 cases, Am J Ophthalmol., 133: 2002; 571-2.

50. Harbor JW, Smiddy WE, Rubasen PE, Murray TG, Davis JL, Flynn HW: Jr. Pars plana vitrectomy for chronic psuedophakic CME, Am J Ophthalmol., 120: 1995; 302-7.

51. Federman JL, Annesley WH, Sarin LK, Remer P: Vitrectomy and CME, Ophthalmology 87: 1980; 622-28.

52. Rosette A, Doro D: Retained intravitreal lens fragments after phacoemulsification, complications and visual outcome in vitrectomised and non vitrectomised eyes, J Cataract Refract Surg.,28: 2002; 310-5.

53. Flach Allan J et al: Improvement in visual acuity in chronic aphakic and psuedophakic CME after treatment with topical 0.5% ketorolac eyedrops, Am J , vol. 112(5): 1991; 514-519.
54. John R Wittpenn & Steven Silverstein: study on topical NSAIDS plus steroids vs topical steroids alone for CME, American journal of ophthalmology vol.146: oct 2008; issue 4
55. Keith A Walter & Amy J Estes: study on management of ocular inflammation following routine cataract surgery, US Opthalmic review, vol.4-2:2011; p.97-100.
56. Miami study group: cystoid macular edema in aphakic and psuedophakic eyes, AJO vol.88(1):july,1979;p.45.
57. David L. Epstein: CME 13 years after cataract extraction, AJO, 83 (4):1977:501.
58. Richard M. Klein & Lawrence Yannuzzi: Cystoid macular edema in first week after cataract extraction, AMJ Ophthalmol,vol. 81(5) :1976; 614.
59. Sanfold L. Severin: late onset CME in pseudophakia, AJO vol.90(2) :aug 1980;p.223-225.
60. Michael S Lee & Jonanthan H Lan: Rapid response of macular edema related to NdYAG capsulotomy to 0.5% ketorolac, Ophthalmic surgery lasers imaging,vol.35(2): Mar – April 2004: p.162-164.

61. Flash Allan J, Dream BJ, Irvine AR: Effectiveness of ketorolac tromethamine 0.5% ophthalmic solution for chronic aphakic and pseudophakic CME ,AJO,103:1987; 479-86.

62. Nagpal M, Nagpal K, Nagpal PN: Post cataract CME, Ophthalmology Clinics Of North America, 14 : dec 2001; p651-659.

63. Rho, David S: Treatment of acute psuedophakic CME with diclofenac vs ketorolac, JCRS, vol 29 (2):dec 2003; p. 2378-84.

64. Chamblers William Stephen: Incidence of CME after phaco, ophthalmology Vol 86(11):nov 1979; p2019-2023).

65. Heler, Jeffrey et al: ketorolac vs prednisolone vs combination therapy in treatment of acute pseudophakic CME, ophthalmology, vol 107 (11):nov 2000; p2034-2038.

66. Allan J.Flash: Incidence, pathogenesis and treatment of cystoid macular edema following cataract surgery, Trans American ophthalmology society-vol 96: 1998; p. 557-634.

67. Iliff W Jackson: Aphakic CME and the operating microscope- is there a connection; Trans American Ophthalmol soc,83: 1985;476-500.

68. Bradford J David ; Wilkinson and Bradford Jr Reagan.H: CME following ECCE with PCIOL, Retina vol 8: 1988; p 161-164.

69. Jampol Lee M:Cystoid macular edema following cataract surgery, Archives of ophthalmology, vol 106:1988; p 894-895.
70. Ursell Paul G et al: CME after phacoemulsification, relationship to blood aqueous barrier damage and visual acuity, JCRS vol 25 (11): 1999 nov; p 1492-1497.
71. Conway Mandi et al: Intravitreal triamcinolone acetonide for refractory chronic pseudophakic CME, JCRS , vol 29:jan 2003; p 27-33.
72. Jost B Jonas ,Kreissig and Robert F: intravitreal injection of triamcinolone acetonide for psuedophakic CME ,AJO, vol 136(2):2003; p 384-386.
73. Linda M Meyer & Carl Ludwig: CME after cataract surgery resolved with intravitreal steroid implant, Case report **o**phthalmol, vol 2 :2011; p. 319-322.

PROFORMA:

Name : Date :

Age : OP/IP no:

Sex : 1. Male ☐ Eye : 1.Right ☐

2.Female 2.Left

Address :

- **Duration from cataract surgery** ☐

  Date of surgery :

  1. 4 – 6 weeks
  2. 7 – 12 weeks
  3. 12 – 24 weeks
  4. > 24 weeks

- **Type of cataract surgery** ☐

  1. ECCE with IOL
  2. SICS with IOL
  3. PHACO with IOL

- **Type of IOL** ☐

  1. PCIOL
  2. ACIOL

- **Preoperative risk factors**

  A. Systemic illness ☐

  1. Diabetes mellitus 2. Hypertension

  3. Both 4. No

  B. Treatment for post operative uveitis ☐

  1. Yes 2. No

  C. Pre-op use of topical NSAIDs ☐

  1. Yes 2. No

  D. CME in other eye ☐

  1. Yes 2. No

- **Intraoperative complications**

  a) PC rupture ☐ 1. Yes 2. No

  b) If yes, Vitreous loss ☐ 1. Yes 2. No

  c) Vitrectomy done ☐ 1. Yes 2. No

d) PCIOL Placement ☐ 1. In bag

2. In sulcus

e) Type of IOL ☐ 1. PCIOL

2. ACIOL

- **Post –op examination**

1. Visual acuity

Unaided

BCVA

Anterior segment examination

2. Uveitis ☐ 1. Yes 2. No
3. Vitritis ☐ 1. Yes 2. No
4. Other findings ☐ 1.Yes 2. No
    a. Iris incarceration in wound
    b. Vitreous incarceration in wound
    c. Vitreous in AC
    d. Peaked pupil
    e. PC rent

Posterior segment examination

Slit lamp examination with 90 D lens

1. CME ☐ 1. Yes 2. No
2. Clinical vitreomacular traction ☐ 1. Yes 2. No
3. Other findings ☐
    1. Epiretinal membrane
    2. Macular hole
    3. Serous retinal detachment
    4. Diabetic retinopathy
    5. Hypertensive retinopathy
    6. Central retinal vein occlusion
    7. Branch retinal vein occlusion
    8. Nil

Treatment given: ☐

1. Topical steroids & topical NSAIDs
2. Periocular steroids
3. Intra vitreal steroids
4. Pars plana vitrectomy

FOLLOW UP

Name : MRD No :

Date :

Vision 1.Unaided

2. Best corrected visual acuity

Anterior segment examination

1. Uveitis : yes /no
2. Vitritis : yes / no

Tension (by applanation tonometer) :

Treatment with timolol : yes / no

Fundus

CME resolved

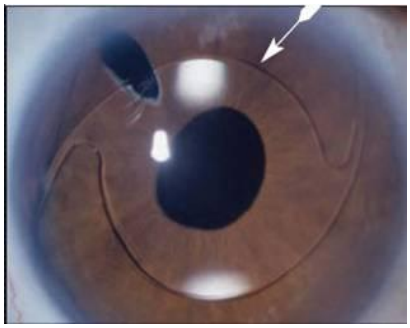
1. Clinically by slit lamp 90 D : yes / no

2. By FFA : yes / no

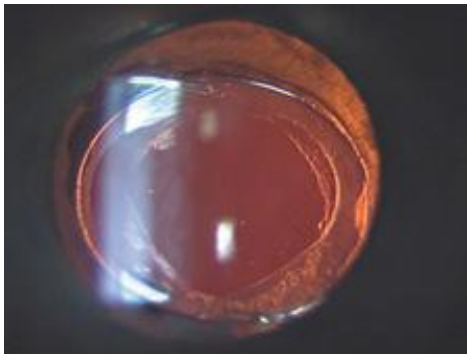
Treatment given

1. Topical NSAIDs
2. Periocular steroids
3. Intra vitreal steroids
4. Pars plana vitrectomy

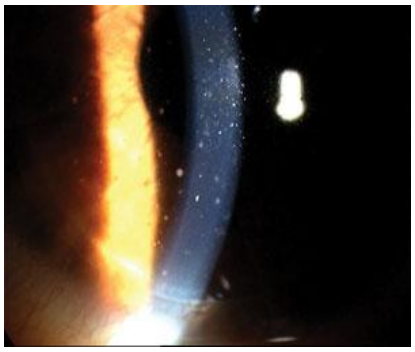
RISK FACTORS FOR CME



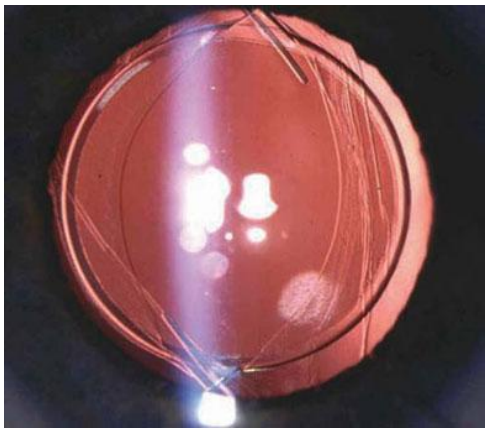
ACIOL



POST Nd YAG CAPSULOTOMY

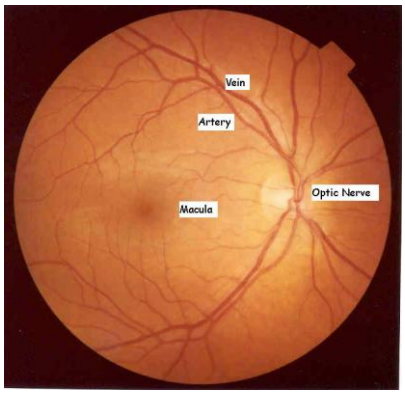


POST OP UVEITIS

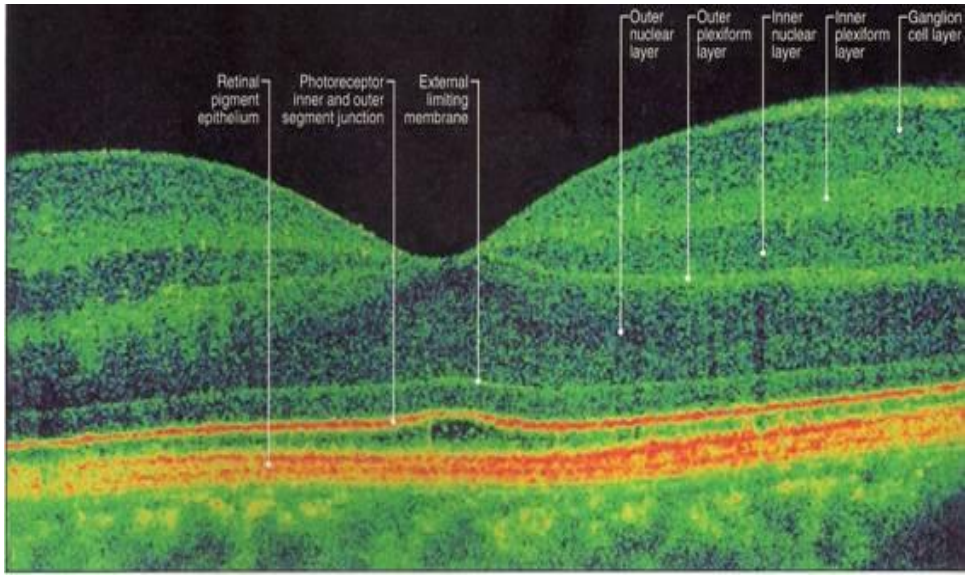


PC RUPTURE

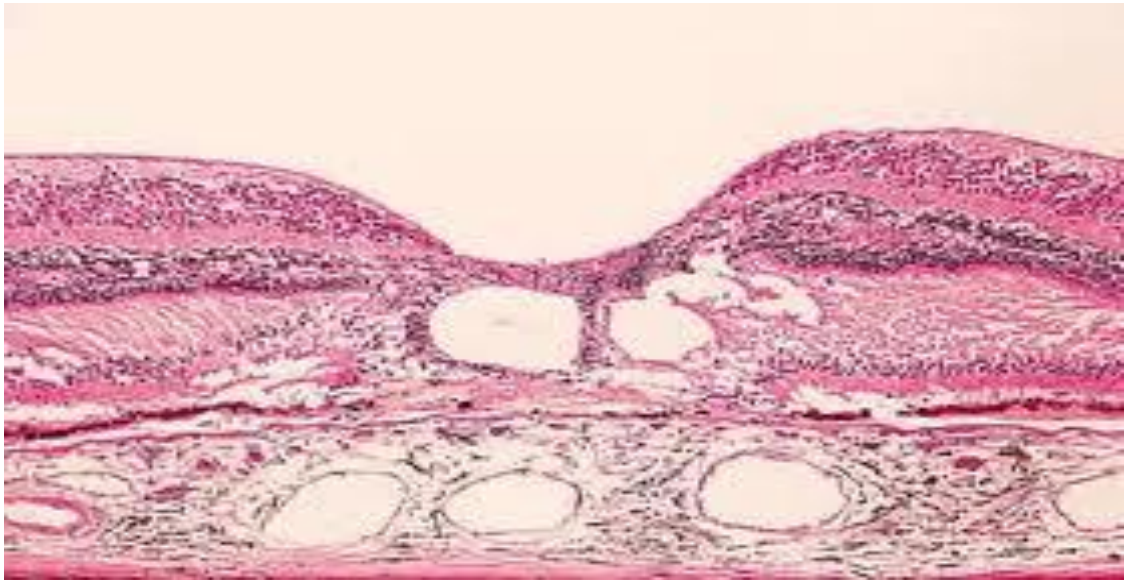
ANATOMY OF RETINA



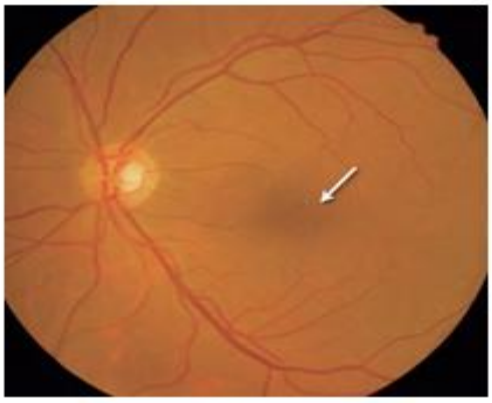
OCT IMAGE OF NORMAL RETINA



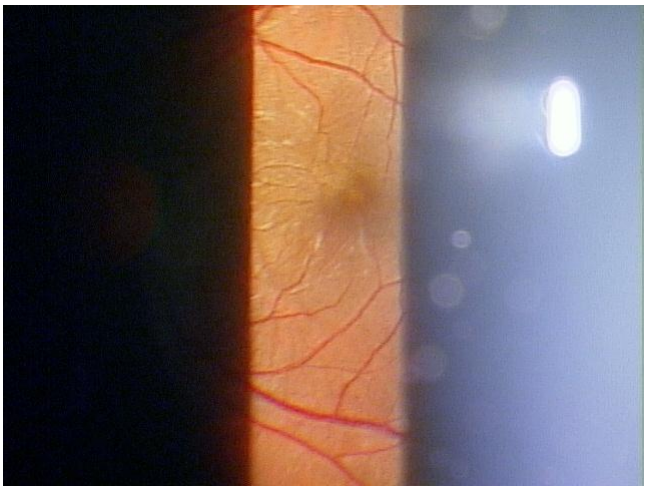
HISTOPATHOLOGY OF CME



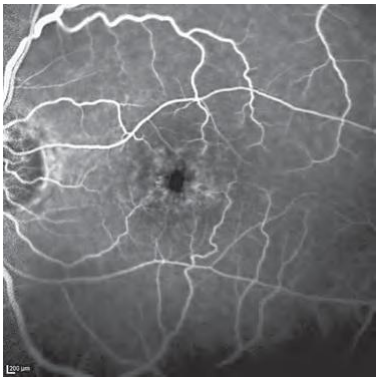
Fundus photo showing CME



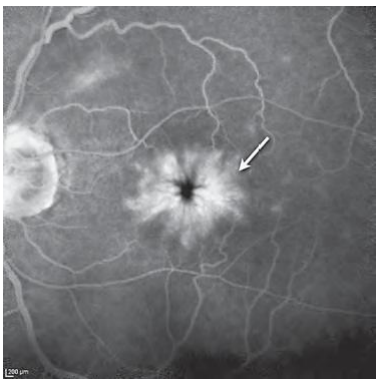
Slit lamp biomicroscopy showing CME



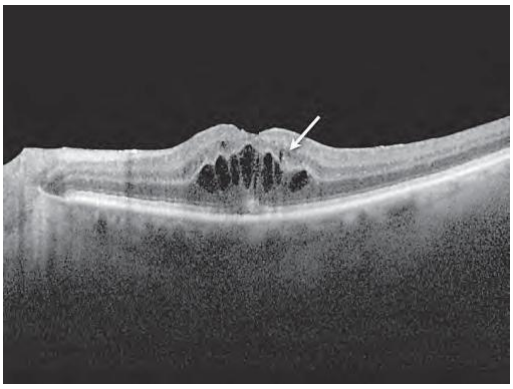
FFA in early phase showing cystoid spaces



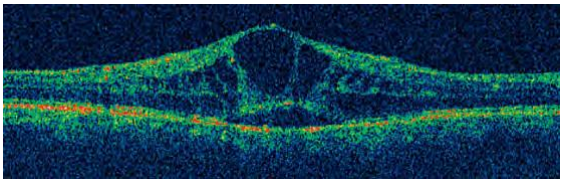
FFA in late phase showing flower petal appearance of CME



Spectral domain OCT showing CME



SD OCT



Cystoid spaces in the outer plexiform layer

Ref. No. 3104/E4/3/2012

Govt.Rajaji Hospital,Madurai.20.
Dated: .03.2012

Institutional Review Board / Independent Ethics Committee.
Dr. A. Edwin Joe, M.D (FM), BL.,
Dean, Madurai Medical College & 2521021 (Secy)
Govt Rajaji Hospital, Madurai 625020.
Convenor
grhethicssecy @gmail.com.

Sub: Establishment-Govt. Rajaji Hospital, aMadurai-20-
Ethics committee-Meeting Agenda-communicated-regarding.

PQR 03/05/12

The Ethics Committee meeting of the Govt. Rajaji Hospital, Madurai was held at 11.00 Am to 1.00Pm on 29.03.2012 at the Dean Chamber, Govt. Rajaji Hospital, Madurai. The following members of the committee have been attended the meeting.

1. Dr.N.Vijayasankaran,M.ch(Uro.) 094-430-58793 0452-2584397	Sr.Consultant Urologist Madurai Kidney Centre, Sivagangai Road,Madurai	Chairman
2. Dr.P.K. Muthu Kumarasamy, M.D., 9843050911	Professor & H.O.D of Medical, Oncology(Retired)	Member Secretary
3. Dr.T.Meena,MD 094-437-74875	Professor of Physiology, Madurai Medical College	Member
4. Dr. S. Thamilarasi, M.D (Pharmacol)	Professor of pharmacology	
5. Dr.Moses K.Daniel MD(Gen.Medicine) 098-421-56066	Professor of Medicine Madurai Medical College	Member
6. Dr.M.Gobinath,MS(Gen.Surgery)	Professor of Surgery Madurai Medical College	Member
7. Dr.S. Dilshadh, MD(O&G) 9894053516	Professor of OP&Gyn Madurai Medical College	Member
8. Dr.S.Vadivel Murugan., M.D, 097-871-50040	Professor of Medicine Madurai Medical College	Member
9. Shri.M.Sridher,B.sc.B.L. 099-949-07400	Advocate, 2, Deputy collectors colony 4th street KK Nagar, Madurai-20.	Member
10. Shri.O.B.D.Bharat,B.sc., 094-437-14162	Businessman Plot No.588, K.K.Nagar,Madurai.20.	Member
11.Shri. S.sivakumar,M.A(Social) Mphil 093-444-84990	Sociologist, Plot No.51 F.F, K.K Nagar, Madurai.	Member

Following Projects were approved by the committee

MASTER CHART

S.No	Patients Name	Age	Sex	IP No	Systemic Disease	Preop BCVA	Postop BCVA	Procedure Done	Risk factors for CME	CME Diagnosed in weeks	Treatment Given	Additional Treatment	BCVA improved after Treatment	BCVA after Treatment	IOP Rise after Steroids	Antiglaucoma Drugs
1	Vinayagam	62	M	81283	No	6/60	6/24	ECCE WITH PCIOL	PCR AND VL	4 to 6	Gp B	Rpt PST	No	6/24	yes	given
2	Rajalakshmi	52	F	81300	No	6/60	6/36	SICS WITH PCIOL	Vitritis	7 to10	Gp B	No	yes	6/12	no	no
3	Chokkalingam	64	M	81365	DM	5/60	6/60	ECCE WITH PCIOL	PCR AND VL	4 to 6	Gp C	No	yes	6/36	no	no
4	Ramasamy	51	M	81378	No	6/36	6/12	PHACO WITH PCIOL	Uveitis	11 to 24	Gp A	No	yes	6/6	no	no
5	Selvaraj	53	M	81465	HT	6/60	6/24	SICS WITH PCIOL	Vitritis	7 to 10	Gp B	No	yes	6/12	no	no
6	Ammapillai	65	F	81488	No	5/60	6/60	ECCE WITH PCIOL	Vitreous in AC	4 to 6	Gp C	No	yes	6/24	no	no
7	Chinnasamy	55	M	81505	No	6/36	6/12	PHACO WITH PCIOL	Uveitis	11 to 24	Gp A	No	yes	6/9	no	no
8	Yasodha	61	F	81512	DM	6/60	6/24	ECCE WITH PCIOL	Uveitis	4 to6	Gp B	No	yes	6/9	no	no
9	Karupasamy	72	M	81289	No	3/60	4/60	E CCE WITH ACIOL	Zonular dialysis	4 to 6	Gp C	No	No	4/60	no	no
10	Saroja	64	F	81536	DM	6/36	6/24	ECCE WITH PCIOL	Vitritis	7 to 10	Gp B	No	yes	6/9	no	no
11	Rasupillai	58	M	81356	No	6/36	6/12	PHACO WITH PCIOL	Uveitis	4 to 6	Gp A	No	yes	6/6	no	no
12	Lakshmi	54	F	81653	No	6/60	6/24	ECCE WITH PCIOL	Vitritis	7 to10	Gp B	No	yes	6/12	no	no
13	Velu	63	M	81699	No	6/60	6/18	SICS WITH PCIOL	Uveitis	7 to10	Gp A	PST inj	yes	6/9	no	no
14	Sundaram	52	M	81680	HT	6/36	6/9	SICS WITH PCIOL	Uveitis	7 to10	Gp A	No	yes	6/6	no	no
15	Shanthi	74	F	81735	No	3/60	6/60	ECCE WITH PCIOL	Uveitis	4 to 6	Gp C	No	yes	6/24	yes	given
16	Raju	67	M	81795	No	6/60	6/24	SICS WITH PCIOL	Vitritis	4 to 6	Gp B	No	yes	6/9	no	no
17	Kamatchi	55	F	81864	No	6/60	6/9	ECCE WITH PCIOL	Uveitis	4 to 6	Gp A	No	yes	6/6	no	no
18	Subbiah	69	M	81899	DM	4/60	6/24	SICS WITH PCIOL	PCR AND VL	4 to 6	Gp B	No	yes	6/12	yes	given
19	Murugan	57	M	82301	No	6/60	6/18	SICS WITH PCIOL	Vitritis	4 to 6	Gp A	PST inj	yes	6/9	no	no
20	Subbamaal	65	F	82355	No	6/60	6/12	ECCE WITH PCIOL	Uveitis	7 to10	Gp A	No	yes	6/6	no	no
21	Marimuthu	71	M	82467	No	5/60	6/60	SICS WITH PCIOL	Vitritis	4 to 6	Gp C	No	yes	6/24	yes	given

22	Kesavan	58	M	82543	HT	6/36	6/18	ECCE WITH PCIOL	Vitritis	7 to10	Gp A	PST inj	yes	6/9	no	no
23	Pitchai	66	M	82678	No	5/60	6/36	PHACO WITH PCIOL	PCR AND VL	4 to 6	Gp B	No	yes	6/12	no	no
24	Vijayalakshmi	54	F	82877	No	6/24	6/9	ECCE WITH PCIOL	Uveitis	11 to 24	Gp A	No	yes	6/6	no	no
25	Rajendran	52	M	82885	HT	6/24	6/12	ECCE WITH PCIOL	Uveitis	11 to 24	Gp A	No	yes	6/9	no	no
26	Sekar	62	M	83193	No	6/60	6/12	SICS WITH PCIOL	Uveitis	7 to10	Gp A	No	yes	6/6	no	no
27	Jebaraj	55	M	83243	No	6/60	6/18	ECCE WITH PCIOL	Vitritis	4 to 6	Gp A	PST inj	yes	6/9	no	no
28	Manikandan	59	M	83367	No	6/36	6/12	SICS WITH PCIOL	Vitritis	4 to 6	Gp A	No	yes	6/9	no	no
29	Maanickam	68	M	83452	No	6/60	6/24	ECCE WITH PCIOL	Vitreous in AC	4 to 6	Gp B	No	yes	6/9	no	no
30	Sundari	49	F	83532	No	6/36	6/12	SICS WITH PCIOL	Uveitis	>24	Gp A	No	yes	6/9	no	no
31	Perumayee	69	F	83566	Both	6/60	6/18	ECCE WITH PCIOL	Uveitis	4 to 6	G p A	PST inj	yes	6/12	yes	given
32	Rengasamy	52	M	83612	No	6/36	6/18	PHACO WITH PCIOL	Vitritis	7 to10	Gp A	PST inj	yes	6/9	yes	given
33	Chinnaperukki	75	F	83722	No	3/60	5/60	ECCE WITH ACIOL	Zonular dialysis	4 to 6	Gp C	PST inj	No	5/60	yes	given
34	Ravi	54	M	83832	No	6/36	6/9	SICS WITH PCIOL	Uveitis	4 to 6	Gp A	No	yes	6/6	no	no
35	Pothumponnu	66	F	83856	No	6/60	6/12	ECCE WITH PCIOL	Uveitis	4 to 6	GpA	No	yes	6/9	no	no
36	Jayachandran	51	M	83934	No	6/24	6/12	PHACO WITH PCIOL	Uveitis	11 to 24	Gp A	No	yes	6/6	no	no
37	Selvam	64	M	83989	Both	6/60	6/36	SICS WITH PCIOL	PCR AND VL	4 to 6	Gp B	Rpt PST	No	6/36	no	no
38	Raasathi	54	F	84134	No	6/24	6/12	PHACO WITH PCIOL	Uveitis	11 to 24	GpA	PST inj	yes	6/6	no	no
39	Palanisamy	62	M	84216	No	6/60	6/24	SICS WITH PCIOL	PCR AND VL	4 to 6	Gp B	Rpt PST	No	6/24	no	no
40	Radhakrishnan	48	M	84278	No	6/36	6/9	PHACO WITH PCIOL	Vitritis	>24	Gp A	No	yes	6/9	no	no

Sl. No	Name of P.G.	Course	Name of the Project	Remarks
1.	Vijayalakshmi. A	PG, M.S (Ophthal)	Clinical study of cystoid macular edema following cataract surgeries.	Approved

Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain Confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution to Government.
2. She/He should inform the institution Ethical Committee in case of any change of study procedure site and investigation or guide.
3. She/He should not deviate for the area of the work for which applied for Ethical clearance. She/He should inform the IEC immediately, in case of any adverse events pr Serious adverse reactions.
4. She/he should abide to the rules and regulations of the institution.
5. She/He should complete the work within the specific period and apply for if any Extension of time is required She should apply for permission again and do the work.
6. She/He should submit the summary of the work to the Ethical Committee on Completion of the work.
7. She/He should not claim any funds from the institution while doing the word or on completion.
8. She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

DEAN

To
All the above members and Head of the Departments concerned.
All the Applicants.